



**Fetal
Alcohol
Syndrome
& Other Alcohol
Related Birth Defects**

AADAC

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Fetal Alcohol Syndrome and Other Alcohol-Related Birth Defects

Second Edition

AADAC

Alberta Alcohol and Drug Abuse Commission
An Agency of the Government of Alberta

Kathy Huebert and Cindy Raftis

1996

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PREFACE


Since 1993, when our original overview of the FAS literature was written, there have been some substantive changes in the area. These changes are reflected in this update.

- One subtle, but significant, shift in recent academic articles concerns fetal alcohol effects (FAE). Several authors have strongly discouraged use of the term FAE. While these articles are not contrary to similar articles published previously, they are sending a much clearer, stronger message that FAE cannot and should not be treated as a diagnosis. Many prominent FAS researchers believe that, at a practical level, it is much more useful to objectively assess and report on specific symptoms or areas of concern for each individual, regardless of whether or not these problems are the result of alcohol use during pregnancy. This more cautious approach is helpful in that it points to specific areas of difficulty and does not subject an individual or parent to the potentially damaging consequences of the label FAE.

In light of the recent recommendations to discontinue use of the vaguely defined term FAE, this review only uses the term “fetal alcohol effects” (FAE) when necessary to directly cite research findings from the literature. When generally discussing the abnormalities caused by alcohol use during pregnancy, the term “alcohol-related birth defects” (ARBDs) is used.

- There is more and better information concerning the long-term effects of FAS into adolescence and adulthood. Persistent behavioural difficulties and emerging psychopathologies are of particular concern in these older patients.

- Paternal alcohol use appears to play a more direct role in causing some problems in offspring than previously thought. Recent animal research suggests that heavy paternal alcohol exposure, even prior to puberty, can result in gender-specific problems in future offspring.
- Though “popular” FAS incidence estimates have not changed since 1993, there has been increasing awareness in the literature of the need to improve FAS surveillance and measurement techniques to better capture the true incidence. In order to better target and evaluate the success of prevention efforts, valid and reliable FAS incidence statistics are essential.
- Although there is still a lack of academic research on effective intervention strategies for those affected by ARBDs, there has been an increase in information on intervention strategies based on parents’ and teachers’ experiences in working with children who have FAS. In general, parents’ and teachers’ experiences emphasize intervention strategies that focus on: (1) environment; (2) learning skills; and (3) consistency.
- As well, prevention of FAS is receiving considerable attention in the current literature. In particular, there has been discussion of various prevention approaches, appropriate targets, and types of prevention strategies such as warning labels for alcoholic beverages and prenatal programs screening for women at risk for having children with alcohol-related birth defects.



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Executive Summary

Since the identification of Fetal Alcohol Syndrome (FAS) in 1973, research on and public interest in the effect of prenatal alcohol consumption on the fetus have steadily increased. AADAC staff regularly receive requests for information and advice about FAS and the effects of alcohol on the fetus. In order to respond to these information needs, a well-researched reference resource on FAS is necessary. This document is an update of the original literature review first written in 1993. As a result of the voluminous research in this area, the overview is not exhaustive. Rather, its purpose is to provide a thorough review of the complex issues surrounding FAS.

Despite the abundant research in the area, there are a number of controversial issues in the FAS literature which make it impossible to easily and concisely state the cause, nature and extent of the problem. Each issue requires that all views in the research and clinical literature be considered. Some particularly contentious issues which emerge in the literature include: ambiguous terminology, difficulties with diagnosis and identification, lack of agreement on a safe level of alcohol consumption, and inadequate information on incidence.

Much of the uncertainty and inconsistencies in the research literature result from the numerous methodological difficulties inherent in conducting FAS research. Some of the research problems that should be considered when interpreting the findings of any study on FAS include: biases in sample selection, limitations of certain research designs (particularly retrospective studies), difficulties in measuring alcohol consumption during pregnancy, and lack of control of possible confounding factors, such as other drug use and home environment,

that may account for the outcome under consideration.

This report is organized according to questions and issues which are commonly raised about FAS. Highlights from the literature pertaining to each of these questions and issues are presented in the remainder of this executive summary.

What Are FAS, Fetal Alcohol Effects, and Alcohol-Related Birth Defects?

Alcohol-related birth defect (ARBD) is a general term used to refer to anatomical or functional abnormalities attributed to prenatal exposure to alcohol. ARBDs occur along a continuum of severity, with miscarriage and stillbirth being the most drastic effects at one end, and various birth defects not sufficient to be classed as FAS at the other end. FAS is a distinct set of anomalies occurring midway along the continuum.

- Fetal Alcohol Syndrome is one type of alcohol-related birth defect describing a specific cluster of abnormalities observed in children born to women with a history of heavy alcohol consumption during pregnancy. The three essential features of FAS include growth retardation, central nervous system damage, and a characteristic face with specific observable traits such as short palpebral fissures (the opening between the eyelids).
 - At the clinical level, there is some debate about the need to confirm the presence of maternal drinking during pregnancy in order to make a diagnosis of FAS. Ideally, knowledge that the mother drank during pregnancy is desirable and adds confidence to the diagnosis. However, it is not always possible
-

to confirm maternal drinking, particularly when a child has been adopted.

- Fetal alcohol effects is an ambiguous term used in several ways. In the literature, it is most commonly used to refer to children who exhibit one or two of the three characteristic features of FAS, with no judgement as to the severity of the effects in relation to FAS. Most prominent researchers and clinicians in this area do not recommend using the term FAE because there are no firm diagnostic criteria for it. Because individual malformations or problems can be caused by a multitude of factors, it is impossible to definitively attribute one or two abnormalities to maternal drinking. There is a reluctance to make such a subjective and uncertain diagnosis because of the problems it may create in terms of people feeling guilty or worrying about having drunk even very small amounts of alcohol during pregnancy. Additionally, the label FAE does not clarify specific areas in which an individual is experiencing problems and is, therefore, not useful in identifying management or referral strategies. Instead of applying the label FAE to a person suspected of having birth defects caused by alcohol, it is much more helpful to caregivers and involved professionals if the specific areas of difficulty are identified. The results of specific physical, psychological and behavioural tests form the best, most objective basis for making decisions concerning special programs or services which the individual patient requires.

How Is FAS Identified?

- FAS is the only alcohol-related birth defect with specific diagnostic criteria.
- An FAS diagnosis can only be made when the patient has signs of abnormality in each of the following three categories:

- (1) prenatal and/or postnatal growth retardation,
- (2) central nervous system impairment, and
- (3) characteristic facial features.

If possible, confirmation of maternal drinking during pregnancy should be obtained.

- Diagnosis of FAS and other alcohol-related birth defects is very difficult for several reasons including:
 - > there is no single symptom peculiar to FAS;
 - > although diagnostic tests are being developed and tested, there are currently no standardized diagnostic tools specific to FAS;
 - > many symptoms vary with age;
 - > infancy is the most difficult time to identify FAS;
 - > it is difficult to apply the diagnostic criteria across cultures as some facial features are normal characteristics for some races and some diagnostic tests (e.g., IQ tests) are biased towards certain segments of the population;
 - > it is difficult to determine whether neurobehavioural problems are due to prenatal alcohol exposure or related to the postnatal environment;
 - > FAS may be overlooked as a diagnosis because it is a relatively newly identified condition; and
 - > it is difficult to measure maternal alcohol abuse.

What Are the Effects of Prenatal Alcohol Exposure?

Overall, findings from studies on the effects of prenatal alcohol exposure vary and depend on the type of effects under study, age of the persons with FAS being studied (e.g., infants, children, adolescents or adults), and the level of maternal alcohol use.

- Problems encountered during the pregnancy or birth itself include: spontaneous abortion, prematurity, breech births, and possibly still-birth.
- Prenatal and postnatal growth retardation.
- Characteristic facial features.
- Central Nervous System (CNS) involvement:
 - > neurological abnormality (e.g., hearing disorders)
 - > developmental delay
 - > behavioural dysfunction or deficit
 - > intellectual impairment (e.g., learning disability, mental retardation) and/or structural abnormalities (e.g., brain malformations).
- Children with FAS are also at higher risk for birth defects that are not necessarily associated with alcohol use; these problems include: abnormalities of the eyes, ears, organs, skeleton, and immune system.
- Long-term effects: small stature and neurobehavioural problems seem to persist throughout the lifespan of the affected person.
- Major psychosocial problems experienced in adolescence and adulthood: attention deficit problems, impulsivity, poor judgement, social-relationship problems, emotional problems, difficulty with organizational skills, difficulty in recognizing and setting boundaries, and abnormally increased and uncontrollable muscle movement (hyperkinesia).
- Behavioural and psychopathological problems, which appear to increase in adolescence, are viewed by many as the most debilitating conditions afflicting these individuals. More research is needed to identify

effective ways to improve the behavioural outcomes of FAS patients.

Is There a Safe Level of Drinking during Pregnancy?

The research findings do not support any “safe” pattern or level of maternal drinking during pregnancy. As well, because alcohol exposure does not benefit the developing fetus and is unnecessary for the mother’s health, most researchers recommend complete abstinence or suggest that “less is better and none is best.” To prevent exposure before pregnancy is diagnosed, most researchers and physicians advise women who are planning a pregnancy abstain from alcohol. This advice is also applied to nursing mothers.

There are five major factors which influence the severity and range of expression of alcohol-related birth defects.

- Quantity of alcohol consumed during pregnancy.
 - > There is general agreement in the literature that the range and severity of anomalies increases with the amount of alcohol consumed during pregnancy.
 - > Not all women who drink heavily during pregnancy will have an FAS child; various authors cite risk rates for FAS among alcoholic women of between 6% and 50%.
- Timing of exposure to alcohol
 - > There is no safe time to consume alcohol during pregnancy. Drinking during the first 12 weeks of gestation may lead to structural and anatomical defects (such as head and facial malformations), while drinking during the second and third trimesters of pregnancy may affect growth and cause functional impairment.
 - > Authors generally concur that full-blown FAS is probably not due to a single binge episode, but

is caused by regular consumption of alcohol throughout gestation. However, episodic binge drinking at high levels is viewed as particularly problematic, resulting in partial expression of the syndrome, with the abnormalities being unique to the period of exposure.

- > Drinking during lactation appears to affect developing infants' brain growth and to reduce infants' milk intake. No safe level has been identified for nursing mothers.
- **Mother's metabolism**
 - > The mother's metabolism influences the extent of damage to the fetus. The length of time the mother has been drinking prior to the pregnancy, her age, body weight, drinking style and the conditions of the pregnancy itself all influence the mother's metabolism.
- **Other factors such as nutrition, smoking, and use of other drugs.**
 - > While the research literature is in agreement that prenatal alcohol intake is the primary cause of FAS, it is much less clear on the role that other suspected prenatal and postnatal risk factors (e.g., nutrition, parenting skills) play in contributing to or exacerbating FAS.
- **Individual response by the fetus.**
 - > Each fetus, being a unique biological system, responds differently to alcohol exposure.

There are several other important issues discussed and debated in the literature regarding threshold levels of alcohol consumption. These issues include the following:

- Researchers now believe that the peak blood alcohol level (as reached during binge drinking) is a better predictor of damage to the fetus than is the duration of exposure or the total amount of alcohol consumed during pregnancy.
- The definition of what constitutes light or moderate versus heavy drinking—many stud-

ies use two drinks per day as the dividing line between “regular” versus “heavy” drinking, but others use three or more drinks per day.

- The level of alcohol consumption shown to cause damage varies greatly from study to study, and partially depends on the particular outcome or effects being investigated.
- Though not all authors agree, many studies acknowledge the possibility that there is a dose-response relationship between alcohol exposure and resulting effects (i.e., that lower levels of alcohol consumption produce milder effects, and that lower levels of exposure are more likely to produce “functional” deficits as opposed to growth problems or malformations). A recent research study showed that even an average of seven drinks per week results in poorer than average mental performance.
- Threshold consumption levels for development of full-blown FAS are not clear. The average daily number of drinks necessary before full-blown FAS can result varies in the literature from two drinks per day to 12 drinks per day.

Can a Father Cause or Contribute to Alcohol-Related Birth Defects?

- One study of humans has found lower birth weights being associated with paternal alcohol exposure during the month of conception. Recent animal research would support that finding as studies have shown offspring are subject to other deficits directly as a result of paternal alcohol exposure, even years prior to conception. In animals, male offspring have altered reproductive

hormonal functioning and spatial learning problems, while female offspring have abnormal levels of stress-related hormones. The underlying mechanisms by which these deficits occur are not yet understood.

- The father can help prevent ARBDs by acting as a role model in reducing consumption and by supporting the woman in reducing her intake.

What Is the Incidence of FAS and Other Alcohol-Related Birth Defects?

- The most widely-quoted estimate of FAS incidence among the general population in the Western world is .33 per 1000 (or one per 3000) live births.
- There are no data available on the incidence of FAS in Canada.
- It is infinitely more difficult to identify children who do not have FAS, but who may have other alcohol-related birth defects. In some of the FAS literature, incidence rates of fetal alcohol effects are estimated at two to three times that for FAS.
- There is general agreement in the literature that most estimates of incidence of FAS are likely to be very conservative.
- The incidence of FAS appears to be higher in certain populations, such as Afro-Americans, Aboriginals and women of lower socioeconomic status. The possible reasons for the higher rates of FAS include genetic or ethnic/racial risk factors and different drinking patterns and norms. As yet, there is no conclusive evidence supporting any of these factors.
- The risk of reoccurrence of FAS appears to be substantially higher for families who already have children affected by FAS as compared to families who do not have offspring with FAS. The estimated risk to younger siblings who already have an older sibling with FAS is 771 per 1000 live births.
- Even though the incidence of FAS is uncertain, some authors have concluded that Fetal Alcohol Syndrome is likely the leading cause of preventable birth defects and is one of the three leading known causes of mental retardation. In certain populations where the incidence of FAS is high, it is the leading cause of mental retardation.

Is There a Concern about Labelling Children as Having Alcohol-Related Birth Defects?

- An early and accurate diagnosis of FAS and other ARBDs is advantageous so that a child's progress can be monitored and appropriate medical, educational, and social interventions made. Another benefit of early identification of at risk children includes the identification and support of women and families potentially at risk.
- A misdiagnosis or unfounded labelling of a child as FAS or other ARBDs can have social and emotional repercussions, result in overlooking other possible conditions, and can unnecessarily burden parents with guilt.
- Diagnosis of FAS can also be a relief for parents as it may explain academic and social difficulties the child is experiencing.
- The benefit of labelling alcohol-related birth defects, such as FAS, in the school setting is debated in the literature. Some researchers feel that the label may unfairly handicap a

child in school. Other people think that knowledge of FAS is critical and can help teachers set realistic performance expectations and tailor their teaching methods.

What Are Some Intervention Strategies for Those Affected by Alcohol-Related Birth Defects?

- It is important to recognize the individual and specific needs of people with ARBDs. The many problems associated with FAS and other ARBDs typically result in the individual having multidisciplinary needs involving the home, the schools, the health care system, vocational training, the criminal justice system, and the community.
- Even though there has not been systematic studies of effective intervention strategies for FAS, parents and teachers have independently developed strategies that have had some positive outcomes in children with FAS. In general, these intervention strategies may be grouped into three categories: (1) environment; (2) learning skills; and (3) consistency.
- Suggested guidelines for parents and others caring for someone affected by ARBDs include:
 - > learn about the signs and symptoms of FAS and other ARBDs and understand that the child has damage to the brain that has medical, social and behavioural consequences;
 - > obtain support in management of hyperactivity and behavioural disturbances;
 - > be an advocate for their child at school and in the community;
 - > prepare to deal with several demanding tasks when their child affected by ARBDs enters adolescence;

- > obtain respite care for themselves; and
- > consider long-term planning for financial and guardianship issues.
- > Guiding principles suggested for educational programs of students who have been prenatally exposed to alcohol or drugs include: early intervention, targeting functional skills, teaching communication skills, and teaching social skills.

What Are Some Strategies to Prevent Alcohol-Related Birth Defects?

- Prevention programs range in the number of components and strategies used. Some typical prevention strategies include: public education (e.g., educational print materials, warning labels), professional education, and treatment services (e.g., screening to identify problem drinkers, outreach).
- The overall goal of ARBD prevention programs is to reduce the number of alcohol-related birth defects. Given the inconsistent research findings on a safe level of alcohol consumption during pregnancy, most prevention programs advise abstinence from alcohol for pregnant women.
- The most commonly used approaches or frameworks in ARBD prevention are the health promotion approach and public health approach. Common to most approaches is that prevention must be: comprehensive; collaborative; community-based; culturally appropriate; nonjudgemental and nonpunitive; and include primary, secondary, and tertiary prevention.
- Singular prevention strategies, such as warning labels, are not effective. A combination of general population and targeted strategies to high risk groups are needed. However, if there are limited resources to spend, they are better targeted at high risk groups, especially women who have already had one child with FAS.

-
- Cooperation and collaboration among many social agencies and community groups allows for a comprehensive range of services responsive to women and their families' needs. It is important to have effective links between health care and addictions treatment services.
 - Research suggests that pregnant women with substance use problems are unlikely to seek treatment due to: the tremendous stigma attached to being pregnant and alcoholic, fear that their newborn baby will be apprehended, lack of support of family and friends for entering and completing treatment, and failure on the part of the treatment system to acknowledge women's needs (e.g., need for childcare).

Conclusion

ARBDs, such as FAS, are very complex and emotional issues. Despite the abundant research on alcohol-related birth defects, several gaps in knowledge and much uncertainty remain. It is not possible to provide straightforward answers to many questions that are commonly asked about alcohol consumption during pregnancy and FAS. Some issues that emerge in the literature as being particularly contentious are:

- the terminology in the area is ambiguous;
- the diagnosis and identification of children with FAS and other ARBDs is very difficult. Because of this fact, children can be labelled incorrectly and caution is urged in making a diagnosis;
- the research findings do not support any safe level or pattern of alcohol consumption during pregnancy; and
- there is inadequate information on the incidence of FAS and other alcohol-related birth defects; differing estimates are found

throughout the literature, and none are available for Canada.

From the literature, it is apparent that ARBDs may be addressed on many different levels; from public education efforts to increase awareness of ARBDs to comprehensive, multi-disciplinary prevention efforts involving several programs and agencies. Because of the tremendous cost and ramifications of ARBDs to those individuals so afflicted as well as to parents and other caregivers, educators, social service professionals, and health care professionals, a balanced and multidisciplinary response to this complex problem is needed.

1. Introduction

As evidenced by considerable media attention, the large volume of publications in the literature, the growing number of workshops and conferences, and information requests to AADAC from allied professionals and parents managing and caring for children with FAS, interest in FAS has escalated since the mid-1980s. Indeed, AADAC offices regularly receive requests for information and advice about alcohol-related birth defects. In order to respond to these information needs, a well-researched reference resource on FAS is necessary. This document is an update of the original literature review first written in 1993. It is not an exhaustive evaluation of the literature. Rather, our goal is to provide a thorough review of the complex issues surrounding FAS.

Many questions have been put forward by AADAC staff. Is it wise for women to drink when pregnant? Is there a minimum safe level? At what stage is drinking most harmful to the baby? How do you identify alcohol-related birth defects? What is the process to diagnose FAS? What is the best way to work with children with FAS? What strategies can caregivers including parents, teachers, and social workers, use to deal with people with alcohol-related birth defects? What is the father's role in causing, contributing, and/or preventing FAS? This report is organized to address these and other specific questions commonly raised about alcohol-related birth defects. Specifically, these questions include the following.

1. What are FAS, fetal alcohol effects and alcohol-related birth defects (ARBDs)?
2. How is FAS identified?
3. What are the effects of prenatal alcohol exposure?
4. Is there a safe level of drinking during pregnancy?
5. Can a father cause or contribute to alcohol-related birth defects?
6. What is the incidence of FAS and other alcohol-related birth defects?
7. Is there a concern about labelling children as having alcohol-related birth defects?
8. What are some intervention strategies for those affected by alcohol-related birth defects?
9. What are some strategies to prevent alcohol-related birth defects?

FAS and other ARBDs are very complicated and emotional multidisciplinary issues. Although research in the area is extensive, a lot of gaps in knowledge and uncertainty remain. As a result, there are no clear cut answers to many questions. The intent of this overview is to present information about the main ARBD issues in a balanced and objective way. Before presenting the various research findings on the main issues, it is helpful to be aware of the research limitations in the area and to know some history of FAS.

ARBDs is a complex research topic. Many of the inconsistencies in research findings relate to various methodological difficulties inherent in conducting this type of research. Some of these difficulties include: biases asso-

ciated with commonly used study designs in this area (e.g., prospective and retrospective studies); difficulties surrounding the measurement of maternal alcohol consumption; difficulties associated with diagnosing FAS; and a lack of control of prenatal and postnatal confounding factors, such as other drug use, smoking, inadequate nutrition, and other environmental factors. (For more detailed information on these methodological limitations, see Appendix A.) It is important to keep these limitations in mind when interpreting research findings in the literature.

In the 1950s and 1960s, it was discovered that the fetus was susceptible to various agents occurring outside a mother's womb. For example, in 1960, thalidomide, a drug to prevent morning sickness during pregnancy, was identified as a substance which caused the limbs of the fetus to be short and malformed (Ottney, 1991). This challenged the prevailing view that the fetus was protected within the womb. At this time, developmental problems in children were generally attributed to factors occurring after birth, such as poor nutrition and disturbed home environments. Alcohol was still not recognized as a harmful agent. In 1968, a French researcher, Lemoine, published a largely unnoticed study that described four distinctive features of children born to alcoholic mothers (Abel, 1990; Ottney, 1991). In 1973, Jones and Smith published two papers describing a similar pattern of malformations in children born to alcoholic mothers. They called the disorder "Fetal Alcohol Syndrome" (Jones & Smith, 1973, p. 999). Shortly thereafter, alcohol was added to the list of several legal drugs known to cause birth defects and thus became a known teratogen¹ (Kearns, 1979).

¹ A teratogen is an agent or influence that causes physical defects in the developing embryo (Miller & Keane, 1972).

2. What Are FAS, Fetal Alcohol Effects, and Alcohol-Related Birth Defects?

Much of the literature in this area focuses on defining terms. Accordingly, this section briefly describes how three terms are used in the literature and in this report.

Fetal Alcohol Syndrome (FAS) is a term used to describe a pattern of abnormalities observed in children born to women with a history of heavy alcohol consumption during pregnancy. The characteristic features include growth retardation, central nervous system (CNS) impairment, and characteristic facial features. FAS is a medical diagnosis that can only be made when a child has signs of abnormalities in each of these three categories (Sokol & Clarren, 1989; Burgess & Streissguth, 1992; Ives, 1987).

Alcohol-related birth defects (ARBDs) is a general term used to refer to anatomical or functional abnormalities attributed to prenatal exposure to alcohol (Clark, 1993; U.S. National Institute on Alcohol Abuse and Alcoholism [NIAAA], 1991; Sokol & Clarren, 1989). There is general agreement that ARBDs occur along a continuum of severity, with miscarriage and stillbirth considered the most drastic effects at the severe end of the continuum (Ottney, 1991). FAS is a specific type of ARBDs that occurs in the middle of the continuum. A variety of birth defects that are not sufficient to be classed as FAS, such as behavioural problems, appear at the other end of the continuum (Clark 1993).

Fetal alcohol effects is an ambiguous term used in several ways. Originally, Clarren and Smith (1978) used the term “suspected fetal alcohol effects” which later was, in the opinion of some authors, misinterpreted and used incorrectly (Clark, 1993; Sokol & Clarren, 1989). Acknowledging the reluctance to positively identify FAS “without some alteration in brain function, growth and facial appearance,” Clarren and Smith (1978, p. 1063) originally stated that “less complete partial expressions can only be referred to as ‘suspected fetal alcohol effects’.” Some interpreted this statement to mean that fetal alcohol effects indicated a birth defect judged milder than FAS, rather than the intended meaning that there was insufficient information to diagnose FAS.

The term fetal alcohol effects is used in the literature in several different ways.

- Fetal alcohol effects as a lesser or milder form of FAS is commonly found in the literature (D’Entremont, 1990; Addiction Research Foundation [ARF], 1992; Burgess & Streissguth, 1992; Canada. Standing Committee..., 1992; Single et al., 1992). However, using the term as a milder form of FAS is inaccurate and can be misleading as some investigators have found that behavioural and functional impairments often associated with fetal alcohol effects can have long-term social consequences just as severe as for those with FAS (Streissguth, 1988).
- The term is often used when a child shows two, but not all three of the characteristic features of FAS (Abel, 1993; Conry, 1990; Ives, 1987; Bauer et al., 1990).
- The term “possible fetal alcohol effect(s)” is used by some individuals in the treatment and prevention community as a basis for obtaining benefits for an individual or in support of public health policy (Sokol and Clarren, 1989; Abel, 1993).

Currently, most researchers and clinicians strongly discourage use of the term "fetal alcohol effects" because there are no firm diagnostic criteria defining it (Aase et al., 1995; Abel, 1993). Many individual symptoms of fetal alcohol effects may be found in children completely independent of maternal alcohol consumption (CCSA, 1994; Abel, 1993). There is also reluctance to make such a subjective and uncertain diagnosis because of the problems that the diagnosis could create (Aase et al., 1995), including:

- assuming that alcohol is the major cause of the child's problem, thus ending the search for other possible causes;
- basing expectations regarding performance in school and at home on that of children with FAS;
- women being stigmatized and feeling guilty about having damaged their children by drinking alcohol during pregnancy, even though in such instances this causation is uncertain;
- physicians becoming frustrated by the imprecision of the "diagnosis," thus not paying attention to any possible contribution of alcohol exposure to their patients' problems; and
- over-identification of fetal alcohol effects that does not help to learn the true magnitude of the problem.

Instead, it is recommended that an objective, individual assessment be conducted to more accurately document specific areas of difficulty (Abel, 1993; Aase et al., 1995; CCSA, 1994). Aase, Jones and Clarren (1995) propose documenting only verifiable conclusions about an individual patient in three areas:

- prenatal alcohol exposure status (i.e., if known or unknown);
- presenting problems (i.e., physical or neurobehavioural problems); and
- diagnostic status (i.e., whether diagnosis is deferred, or cannot be made due to inadequate evidence of maternal alcohol consumption).

Documentation of this type of objective information is much more helpful to physicians, caregivers, and teachers, etc., when making decisions concerning management and referral strategies for an individual.

Recognizing the difficulties involved in applying the term fetal alcohol effects, this report only uses the term if it was included in the original research or literature being cited. However, irrespective of the terminology issue, it is important to acknowledge awareness and concern about the many alcohol-related birth defects that do not meet the full criteria necessary to qualify for an FAS diagnosis (CCSA, 1994).

3. How Is FAS Identified?

FAS is a medical diagnosis usually made by a physician who is experienced in diagnosing FAS. At present, there are no specific criteria to diagnose other alcohol-related birth defects. With FAS, a positive diagnosis is made on the basis of the presence of abnormalities in each of three categories as specified in a standardized set of diagnostic criteria. The diagnostic criteria for FAS, as well as proposed amendments to the criteria and the difficulties associated with making a diagnosis, are detailed in this section.

3.1 Diagnostic Criteria

For several years after the term Fetal Alcohol Syndrome was coined, researchers and clinicians analyzing and describing the impact of alcohol consumption by the mother on offspring used various inconsistent definitions and terms. This confusion led the Fetal Alcohol Study Group of the Research Society on Alcoholism to publish a standardized definition for Fetal Alcohol Syndrome in 1980. In 1987, this same group reviewed the definition of FAS and related terms with the goal of providing “a set of guidelines for use by investigators, care providers, and others that will enhance comparability of results of clinical observations, scientific studies and public health reporting” (Sokol & Clarren, 1989, p. 597). However, consensus could not be reached by the group and no official changes were made to the 1980 definition. In 1989, Sokol and Clarren and several members of the FAS Study Group recommended that only minor modifications be made to the guidelines published in 1980, and that the following criteria for diagnosis of Fetal Alcohol Syndrome supersede those originally published (Sokol & Clarren, 1989).

The diagnosis of FAS can only be made when the patient has signs of abnormality in each of the following three categories:

- prenatal and/or postnatal growth retardation (weight and/or length or height below the tenth percentile when corrected for gestational age (i.e., age of the baby in weeks since conception);
- central nervous system (CNS) involvement (including neurological abnormality, developmental delay, behavioural dysfunction or deficit, intellectual impairment and/or structural abnormalities such as microcephaly (head circumference below the third percentile) or brain malformations found on imaging studies or autopsy); and
- a characteristic face, qualitatively described as including short palpebral fissures (the opening between the eyelids), an elongated midface, a long and flattened philtrum (the vertical groove above the upper lip), thin upper lip, and flattened maxilla (bones forming the upper jaw or midface).

These three general criteria are the minimum set of criteria which must be met in order to make a medical diagnosis of FAS. Based on this definition, a diagnosis of Fetal Alcohol Syndrome could be based purely on physical examination of the offspring—this is important as many FAS children are given up for adoption and the birth mother’s drinking behaviour can not be able to be ascertained. However, Sokol and Clarren (1989, p. 598) point out that “clearly, a relationship between the prenatal impact of alcohol on the offspring should be sought and reported.” In lieu of establishing a history of maternal drinking during pregnancy, some researchers consider the presence of a sibling with FAS to constitute proof that there is a history of maternal drinking (Ives, 1987; Robinson et al., 1987; Robinson et al., 1992).

3.2 Difficulties Diagnosing Fetal Alcohol Syndrome

There are many difficulties in diagnosing Fetal Alcohol Syndrome. Firstly, no single symptom is specifically distinctive or characteristic to FAS (Abel, 1984 as cited in Abel, 1990; Ernhart, 1991) and there are no standardized psychological or behavioural tools which can be used for a definitive diagnosis (Burgess & Streissguth, 1992). Day and Richardson (1991) point out that there is even a broad spectrum of severity among diagnosed FAS patients with respect to motor and speech development and behavioural problems.

Because of the difficulties in diagnosing FAS, weighted checklists of distinguishing features are being developed and tested. In the future, it is likely that technology which aids in the detection and diagnosis of disorders involving brain dysfunction (such as magnetic resonance imaging (MRI)) will be applied in the study of children with FAS (Aase, 1994). One recent study that used MRI to examine the brains of 13 children with FAS, found selective reductions in the size of certain regions of the corpus callosum in all of the children (Riley et al., 1995).

Age-related changes in the appearance of various symptoms also makes diagnosis difficult and, in fact, raises questions about whether or not a syndrome actually exists. In some instances, effects appear later or may even disappear with age (Abel, 1990). It is generally agreed that the neonatal period is the most difficult time to recognize FAS and that a majority of affected individuals are likely to be identified at older ages (Sokol & Clarren, 1989).

Central nervous system impairments which are manifested as neurobehavioural effects are difficult to establish in the newborn (Abel, 1990). Frequently, neurobehavioural problems, such as hyperactivity, impulsivity, attention deficits, and memory and learning problems, are first identified when the child reaches school age (Streissguth, 1991). Further, conditions which may appear in the newborn to be symptoms of CNS impairment are often actually symptoms of alcohol withdrawal in the baby which do not necessarily predict long-term neurological deficits (Coles et al., 1987).

The specific craniofacial features needed to positively identify FAS are also difficult to recognize, particularly in the newborn period. In a clinical manual of substance abuse, Streissguth (1991) has suggested that facial and growth characteristics are most noticable at between six months and three years of age. Usually, experienced dysmorphologists can accurately make the diagnosis of FAS.

Application of the standard diagnostic criteria across cultures may also be problematic. For instance, Native Americans have a genetic trait for epicanthic folds (vertical folds on either side of the nose, near the inner eye) and Afro-Americans have a significantly different palpebral fissure size (slit or opening between the eyelids) than Caucasians (Fuchs et al., 1980 as cited in Abel & Sokol, 1991). Robinson and associates (1987) reported that the facial characteristics of Indian children in their study made analysis of facial characteristics a more subjective rather than objective process. They questioned the validity of using facial characteristics to diagnose these children as having FAS or fetal alcohol effects, and believed their caution resulted in underdiagnosis of FAS and fetal alcohol effects in their study. However, if

less caution were used, overdiagnosis could conceivably result. As well, the researchers express caution in administering and interpreting Intelligence Quotient (IQ) tests which are used to evaluate CNS dysfunction because often performance IQ is significantly higher than verbal IQ among Native children (Robinson et al., 1987). Despite this caution, however, Robinson and associates actually found that the Native children they studied with FAS and fetal alcohol effects had both verbal and performance IQs which were at a borderline retarded level with no difference between the two types of abilities.

Another difficulty in making a conclusive diagnosis of FAS is that neurobehavioural effects are affected by postnatal experience, and it is very difficult to conclusively attribute the cause of such problems to alcohol exposure during gestation.

Diagnosis may also be overlooked because FAS is a relatively newly identified condition (Roman et al., 1988). Physicians are not equally skilled in identifying the anomalies associated with FAS (Abel & Sokol, 1991). Giunta and Streissguth (1988, pp. 453-454) state that "although diagnosis of FAS should be made by a dysmorphologist, geneticist, or knowledgeable physician, identification of children possibly affected by prenatal alcohol exposure can be carried out by professionals involved in service delivery. Otherwise, many patients' needs may go unmet due to lack of identification."

Finally, a major diagnostic problem concerns the ascertainment of maternal alcohol abuse. Problem drinkers tend to under-report drinking behaviour during pregnancy due to denial or poor recall (Sokol & Abel, 1992).

4. What Are the Effects of Prenatal Alcohol Exposure?

It is generally accepted in the literature that alcohol-related birth defects occur along a continuum of severity, with miscarriage and stillbirth considered the most drastic effects at the severe end of the continuum (Ottney, 1991). “On the less severe end of the spectrum are various birth defects not sufficient to be classed as FAS, including both cognitive and behavioural abnormalities” (Clark, 1993, p. 2). FAS is viewed as a well-defined cluster of abnormalities somewhere in the middle of this continuum of alcohol-related birth defects. In general, one or some combination of physical malformations, central nervous system anomalies, and growth deficits are the types of effects focused on in studies of maternal alcohol consumption (Little & Wendt, 1991).

The type and severity of alcohol-related birth defects seen in children who were prenatally exposed to alcohol varies and is thought to depend on the quantity of alcohol consumed, the stage(s) during the pregnancy when the alcohol was consumed, the mother’s metabolism, and the fetus’ genetic makeup (Sokol & Clarren, 1989). When conducting research to ascertain a cause and effect relationship between maternal alcohol consumption and long-term outcome, follow-up studies occurring over several years must consider environmental factors that may contribute to the effects observed in children. It is difficult to sort out whether the cause of the observed effects are due to prenatal factors, postnatal factors, or both (Streissguth, 1986).

Overall, study findings of the effects of prenatal alcohol exposure vary and depend on the type of effects under study, age of the persons being studied (e.g., infants, children or adolescents), and the level of maternal alcohol use.

Detailed information regarding the physical effects of maternal alcohol consumption, before birth and during infancy, are presented in Appendix B. Both the range of problems caused by prenatal alcohol exposure and more common findings are outlined below. The effects are described chronologically from before birth to adolescence and young adulthood.

4.1 Physical Effects During Pregnancy, Birth, and Infancy

As detailed previously in the section entitled “Diagnostic Criteria,” FAS is presently diagnosed on the basis of three minimum criteria—prenatal and postnatal growth retardation, evidence of central nervous system involvement, and characteristic facial features. However, many clinicians and researchers have frequently noted other abnormalities in patients with FAS. Based on a comprehensive review of case study and epidemiological literature, Abel (1990) has summarized the frequency of occurrence of various types of alcohol-related birth defects. Problems encountered during the pregnancy or birth itself which are thought to be caused by maternal alcohol consumption include:

- spontaneous abortion;
- prematurity;
- breech births; and
- possibly stillbirth.

Children with FAS are also at higher risk for birth defects that are not necessarily associat-

ed with alcohol use (Abel, 1990; Aase, 1994). None of these problems are specific to FAS, therefore their presence can only help support, not define, a diagnosis of FAS. These problems include:

- eye abnormalities;
- ear abnormalities;
- various organ pathology (e.g., congenital heart problems, urinary tract and genital anomalies);
- skeletal defects (i.e., of limbs and joints);
- neural tube defects (e.g., spina bifida);
- immunological problems;
- obstruction of upper airway passages;
- neuropathology; and
- tumors.

Behavioural problems commonly seen in infants with FAS include (Davis, 1992):

- irritability;
- poor sucking ability;
- low tolerance for stimulation; and
- slow development.

4.2 Effects in Preschool Children

Follow-up studies have been conducted on children at four years of age. Researchers have found lower IQ, poor attention, longer reaction time, and other impaired behaviours in those children exposed to alcohol prenatally (Landesman-Dwyer et al., 1981; Coles et al., 1991). As well, reduced growth in weight, height, and head circumference has been found (Steinhausen, Nestler, & Spohr, 1982 as cited in Streissguth, 1986). Other physical and behavioural problems that have been observed include (Streissguth, 1986; Aase, 1994; Steinhausen et al., 1993, 1994 as cited in Streissguth, 1994):

- failure to thrive;
- retarded motor development;
- irritability;
- hyperactivity;
- sleeping and eating problems;
- urinary incontinence;
- speech impairment;
- poor fine motor coordination;
- clumsiness;
- difficulties with peers; and
- hearing problems.

4.3 Effects in School-age Children

Alcohol consumption during pregnancy can cause brain damage, resulting in a wide variety of neurobehavioural and developmental effects on children including intellectual impairment, learning problems, attention and memory problems, fine and gross motor problems, and difficulty with organization and problem solving (Streissguth et al., 1989). School-aged children with ARBDs have been found to display attention deficits and behavioural problems similar to those of children with attention deficit disorder, although children with ARBDs have been found to be more intellectually impaired (Nanson & Hiscock, 1990 as cited in Olson et al., 1992). Steinhausen et al. (1993, 1994 as cited in Streissguth, 1994) identified speech disorders, emotional disorders, unusual habits and stereotypic behaviour in 45%-50% of a sample of FAS children.

In the book *Fantastic Antone Succeeds!* by Kleinfeld & Wescott (1993), Barbara Morse has written a chapter discussing the information processing problems associated with the behavioural disorders of FAS. It is her belief that specific areas of the brain are damaged during prenatal development, causing disturbed behaviours in children with FAS. Based on her research and interaction with children with FAS in education programs, she believes that these kids have severe information processing deficits. Information processing deficits comprise problems in four areas: information input, integration, memory, and output. "Input represents the recording of information from the senses. Memory represents the storage of information for later use. Integration is the process of interpreting the input. Output requires appropriate use of language and motor

skills" (Morse, 1993, p. 32). In the classroom, information processing difficulties may be evidenced by a child who:

- appears to know something one day, forgets it the next, and then knows it again after several more days (possibly because they have lost the ability to retrieve information);
- repeats exactly what they were asked to do, but still does not complete the task (may be due to their expressive verbal skills being better than their receptive skills);
- has difficulty doing anything that involves arranging, sequencing, or taking turns (because they often cannot understand the concept of order);
- repeats words, questions, or actions over and over (because their brain may be overloaded or has difficulty processing information).

Recognition that information processing deficits may be at the root of neuro-behavioural problems in children with FAS may help therapists understand these problems and begin to develop appropriate therapeutic strategies. Certainly, this theory suggests a shift in thinking about children with FAS. For example, "the concept of a naughty, willful child diminishes. Instead, children are seen as misinterpreting what they hear or only being able to process certain pieces of information. They try to make sense out of what their brain is telling them, but that seldom makes sense to the rest of the world" (Morse, 1993, p. 34).

The average IQ scores of children with FAS is 70 (considered mildly retarded), although they vary considerably from severely retarded to normal levels (Landesman-Dwyer, 1982, & Streissguth et al., 1978 as cited in Day and Richardson, 1991). IQ scores are generally stable over time, though some improvement may occur in children with borderline mental retar-

dation (Abel, 1990). No significant differences in IQs have been noted between children raised in foster homes as compared to those raised in their biological homes (Jones, 1986; Phillipson, 1988; Ottney, 1991).

4.4 Effects in Adolescents and Young Adults

Information on the manifestation of FAS in adolescents and adults has been available only recently as longitudinal study data on the first individuals diagnosed with FAS accumulates. It appears that as individuals with FAS grow older, their physical characteristics change considerably; this is a major reason initial identification of FAS is more difficult in older patients (Streissguth, 1994). In particular, several studies have noted elongated faces with longer noses and bigger chins which contribute to coarser looking facial features after puberty. As well, after puberty, girls in particular tend to put on weight, changing their weight to height ratio from a pre-puberty low ratio to a high ratio after puberty.

Unfortunately, longitudinal studies are finding that the central nervous system effects seen in patients diagnosed with FAS appear to be long term (Streissguth, 1994). For example, a follow-up study of 61 adolescent and adult patients who had previously been diagnosed as having FAS or possible fetal alcohol effects, found that:

The average IQ was 68, but the range of IQ scores widely varied. Average academic functioning was at the second-to fourth-grade levels, with arithmetic deficits most characteristic. Maladaptive behaviors such as poor judgment, distractibility and difficulty perceiving social cues were common. Family environments were remarkably unstable. Fetal alcohol syndrome is not just a

childhood disorder; there is a predictable long-term progression of the disorder into adulthood, in which maladaptive behaviors present the greatest challenge to management (Streissguth et al., 1991, p. 1961).

Several other follow-up studies concur that IQ tends to remain stable over time and does not usually improve even when the teen or adult is placed in an improved educational or living situation (Streissguth, 1994).

LaDue et al. (1992) describe the psychiatric and social implications of adolescents and adults with FAS and fetal alcohol effects. The most obvious problems experienced include impulsivity, poor judgement, poor social skills, difficulty in organizational skills, and difficulty in recognizing and setting boundaries. In day-to-day living, those in tightly structured homes with vigilant parents were able to minimize, but not prevent or eradicate problems. Some of the older patients studied were living in shelters or low income housing. Others wandered from relative to relative, and still others were in dysfunctional and/or alcoholic homes. These older patients had difficulty communicating their needs, being self-sufficient, maintaining their own hygiene, and applying for and receiving social services. Many had a great deal of difficulty relating to their age mates appropriately. Often they were the centre of jokes at school, were easily led and manipulated by others, and, as adults, were frequently at risk for social, sexual, and financial exploitation.

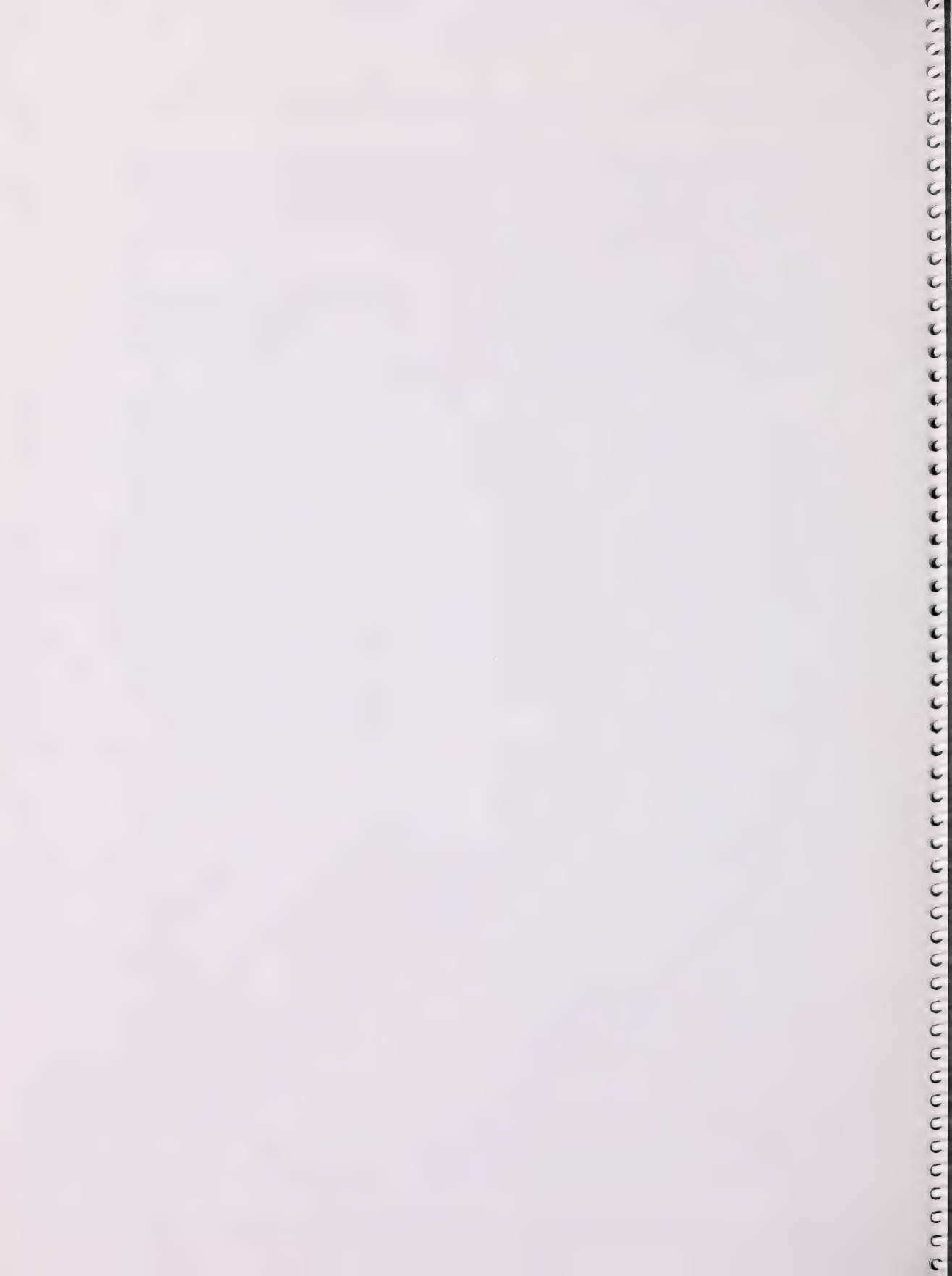
The behavioural problems which accompany FAS are viewed by many as the most debilitating condition afflicting these individuals. In Streissguth and associates' study (1991) of 61 adolescents and adults previously diagnosed as having FAS or possible fetal alcohol effects, they found that 62% of these patients had significant levels of maladaptive behaviours

(including attention deficits, poor judgment, lower comprehension and abstraction abilities as well as conduct problems such as lying and defiance). Comparatively, only 15%-20% of adolescents with Down's Syndrome display these types of behaviours (Harris, 1988 as cited in Streissguth, 1991). It should be cautioned, however, that it is difficult to assess the role that postnatal environmental factors play in development of such behavioural problems.

Recent longitudinal studies of FAS patients by Steinhausen et al. (1993, 1994 as cited in Streissguth, 1994), have found that in the late school years (i.e., kids aged 13 years and older) emotional disorders, followed by hyperkinetic disorders (characterized by abnormally increased and uncontrollable muscle movements) were found in half of the patients. Conduct disorders persisted in 20% of these adolescents. Additional tests of psychiatric symptomatology revealed attention-deficit problems to be most common, followed by social relationship problems.

In considering the results of these various longitudinal studies of teens and adults with FAS, Streissguth (1994) concludes that, "No longer can FAS be viewed as just another type of mental retardation. Not only are there many patients with FAS whose intellectual abilities fall well within the normal range, but they also are displaying an increasing and unsettling degree of recognizable psychopathology" (p. 79). These secondary psychopathologies and disabilities appear in adolescence when it becomes more apparent to the adolescent with FAS that they do not "think quite like everybody else" (Streissguth, 1994, p. 79). The reason for these behavioural difficulties remains unknown, though it appears that they cannot be easily accounted for by the patients' intellectual abilities or the status of their living situ-

ations. Further research is required to identify more effective ways to modify and improve the behavioural outcomes of FAS patients.



5. Is There a Safe Level of Drinking During Pregnancy?

Not all women who drink during pregnancy produce children with Fetal Alcohol Syndrome. The purported risk of an alcoholic woman producing an FAS child varies considerably from study to study. For example, based on a review of a number of retrospective and prospective studies, Jones (1986) pegged the incidence of FAS in children born to alcoholic women at between 30%-50%. Burgess and Streissguth (1992) similarly conclude that approximately 30%-40% of all children born to alcoholic mothers will have FAS. However, other credible researchers in the area cite much lower incidence rates for alcoholic women, for example 6% (Day & Richardson, 1991), and 7.1% (Abel, 1993).

The risk of reoccurrence of FAS within families appears to be substantially higher. In examining the clinical case study literature, Abel (1990) arrived at the following rates of incidence or risk for siblings: (1) the probability of an older sibling having FAS if there was a younger sibling with FAS is 170.4 per 1000 live births; and (2) the risk is substantially higher for younger siblings who already have an older sibling with FAS, at 771 per 1000 live births.

Sokol and Clarren (1989) cite five factors which account for the various forms and intensities of alcohol-related birth defects:

- the quantity of alcohol consumed during pregnancy;
- the gestational timing of consumption;
- the mother's ability to metabolize alcohol;
- other factors such as nutrition, smoking; and other drug use; and
- individual fetal susceptibility/resistance as determined by genetics.

This section details what the research literature says about the role of each of the first four factors in causing FAS and other alcohol-related birth defects (for background information regarding how alcohol reaches and is processed by the developing fetus, please refer to Appendix C).

5.1 Amount of Alcohol Consumed

The subject of safe drinking levels inspires much debate. One of the issues involved when addressing safe consumption levels concerns whether the total amount of alcohol consumed during pregnancy (usually expressed as an average number of drinks per day) is the determining factor in FAS, or whether the peak blood alcohol level is a better predictor of damage to the fetus. Animal models indicate that high blood alcohol levels (like those obtained during binge drinking) predict alcohol-related birth defects better than the total volume of alcohol consumed (Russell, 1991). Streissguth et al.'s (1989) analyses of maternal drinking patterns also supports the importance of binge drinking. As well, drinking prior to recognition of pregnancy was important in their analyses.

A related point of confusion in the literature is the definition of what constitutes light or moderate versus heavy drinking. Various clinicians and researchers have defined these consumption levels differently, making comparison of study results and establishment of threshold levels very difficult. In reviewing the literature, Little and Wendt (1991) found that 30 millilitres of ethanol daily (or about two drinks per day)

is sometimes used as the dividing line for "regular" versus "heavy" drinking. Some authors who have used the frequency of occurrence of heavy drinking episodes during pregnancy in analyzing the effects of consumption on pregnancy outcome have defined a heavy drinking episode as five or more drinks on one occasion (Olson et al., 1992; Day et al., 1991; Streissguth et al., 1989).

Finally, many different types of alcohol-related birth defects are studied by various researchers; depending on the particular effect under study (e.g., birth weight, craniofacial effects, neurobehavioural anomalies, etc.), different levels of consumption have been shown to cause damage. Many authors acknowledge the possibility of a dose-response relationship between alcohol exposure and effects (Little & Wendt, 1991; Olson et al., 1992; Streissguth et al., 1989). In other words, the lower levels of prenatal alcohol exposure are more likely to produce milder effects on the body formation, growth, and/or behaviour of the child (Olson et al., 1992; Streissguth et al., 1989). For example, in a study of classroom behaviour achievement of 11 year olds, Olson et al. (1992) found that as the level of prenatal alcohol consumption increased, child performance decreased and there was no threshold or number of drinks below which no effects were observed. The dose-response relationship typical of most teratogenic substances also finds that lower levels of exposure are more likely to produce "functional" deficits (e.g., learning and behavioural difficulties) than growth problems or malformations (Voorhees & Mollnow, 1987 as cited in Olson, 1992).

In 1992, Levin and associates (as cited in Jacobson and Jacobson, 1994), noted that because of advancements in technology, structural changes in the brain can now be detected

at even lower exposure levels than neurobehavioural deficits. So, it is possible that future studies will show that even low levels of alcohol consumption may cause structural damage insufficient to cause obvious effects on function. There is some concern that unobservable structural damage could lead to functional problems when a child is stressed or challenged, or when the individual reaches old age (Riley, 1990 as cited in Jacobson and Jacobson, 1994).

The case for establishment of a threshold consumption level for development of full-blown FAS is also not clear. Streissguth and associates (1991) state that no child has yet been identified with FAS without a history of chronic exposure to alcohol in utero. Abel (1990) concludes that a woman most at risk for having an FAS child appears to drink an average of ten or more drinks per day. Ottney (1991) states that all FAS cases reported so far have involved alcoholic women drinking more than 12 drinks per day. The Addiction Research Foundation (1992) writes that the consumption threshold for development of FAS is six drinks per day during the first trimester of pregnancy. Casiro (1992) points out that FAS has only been noted in infants of mothers who drank at least two to four drinks daily for prolonged periods. A recent study by Ernhart and associates (1992) concluded that a threshold of three drinks per day during the embryonic period (two to eight weeks) was necessary before craniofacial anomalies consistent with FAS were evident in the newborn (it should be noted that this study offered some reassurance to those women who had an occasional drink early in pregnancy).

Sokol and Abel (1992) caution that estimates of drinking thresholds must be tempered by the awareness that women who drink heavi-

ly may deny and underreport the amount they drink while pregnant. "Consequently, the risk to the fetus of what might appear to be 'two drinks a day' is probably the result of much higher intake" (Sokol & Abel, 1992, p. 95). As well, it is important to consider that seemingly "low" average daily drinking amounts may be the result of one or two heavy drinking occasions per week (Abel, 1993).

There does appear to be general agreement in the literature that the range and severity of anomalies increase with the amount of alcohol consumed. Generally, the findings suggest that heavy or alcoholic drinking by the mother affects the growth, morphology, central nervous system, and a host of other indicators of fetal health (Little & Wendt, 1991; Abel, 1993). There is also extensive evidence that moderate drinking during pregnancy is harmful, but there is a greater proportion of conflicting findings than for studies that focus on heavy alcohol exposure (Little & Wendt, 1991).

Thus far, a critical threshold level of consumption has not been demonstrated. There is likely no single dose-response relationship that can be described as causing FAS or various other alcohol-related birth defects. In 1987, Clarren and Bowden suggested that each CNS, morphological and growth defect has its own dose-response and gestational timing relationship (as cited in Secretary of Health..., 1990). Also, in an article entitled "Prenatal Alcohol Exposure and Neurobehavioral Development: Where is the Threshold?", Jacobson and Jacobson (1994) point out that: "because the threshold values derived from human studies are based on group averages, it is not appropriate to infer that exposure just below a threshold level is necessarily 'safe,' because some individuals could be markedly more sensitive than others" (p. 35).

In conclusion, the research findings do not support any "safe" pattern or level of maternal drinking during pregnancy (Olson et al., 1992). As well, because alcohol exposure has no apparent benefit for the developing fetus, and is not necessary for the health of the mother, most researchers recommend complete abstinence or, suggest that "less is better and none is best" (Addiction Research Foundation, 1992, p. 4). To prevent exposure of the eggs before pregnancy and of the fetus during a critical period in early development that occurs before pregnancy is diagnosed, women who are planning a pregnancy have also been advised to abstain from alcohol as are nursing mothers (American Society of Addiction Medicine (ASAM), 1992).

5.2 Gestational Timing of Consumption

Abel, 1981 (as cited in Overholser, 1990), reports that anatomical malformations primarily occur during the first twelve weeks, but that reduced growth and functional impairment may result from maternal drinking after this point.

Overholser (1990) identifies five critical periods during fetal development when maternal alcohol consumption could affect normal development. The first critical period is at or before conception. Most researchers agree that paternal alcoholism likely damages the sperm and reduces the probability that offspring will be produced. Though very little has been written about the effect of alcohol on the egg, studies done by Kaufman, a world expert on embryology, have shown that alcohol can damage eggs in the ovary before conception. He believes that genetic damage of the egg prior to ovulation may be more serious than the problem of alcohol consumption during the pregnancy itself (Phillipson, 1988).

The second critical period occurs during the first three weeks following conception. This is the early embryonic period and is the time at which development starts and allows for neural tube elaboration. Damage during this period often results in the death of the embryo and abortion (Samson & Diaz, 1982 as cited in Overholser, 1990).

The embryonic period is the third critical period and occurs up to the end of the twelfth week. This is the critical period for development of structural problems and abnormalities in appearance and form. The craniofacial effects characteristic of full-blown FAS are attributed to drinking during this time period (Coles, 1994). Alcohol consumption at this time also appears to diminish the number of brain cells and alters their migration, resulting in mental retardation, microcephaly (small head size) and various congenital malformations (Samson & Diaz, 1982 as cited in Overholser, 1990; Coles, 1994). The decreased number of cells in other parts of the fetus may account for low birthweight and continuing growth retardation seen in FAS children (Ottney, 1991). It is often stated that the most damage to the fetus occurs during the first trimester when organogenesis² is taking place and the fetus is most susceptible to toxins (Streissguth et al., 1989; Addiction Research Foundation, 1992). Gross anatomical malformations are unlikely to occur after the first 12 weeks (Overholser, 1990).

The second and third trimesters comprise the fourth critical period which is primarily a time of fetal growth. Umbreit and Ostrow (1982) (as cited in Overholser, 1990) found that only one ounce of alcohol per day consumed only during the third trimester caused lower birth weights without any of the other FAS symptoms. This is also the period of greatest brain and central nervous system development and

when they are most susceptible to injury (Ottney, 1991). So, whereas drinking during early pregnancy may cause structural brain damage, drinking late in pregnancy may result in developmental delay (Ottney, 1991).

The fifth and final period during which fetal alcohol effects can occur is during postnatal development; the infant's brain and central nervous system are continuing to develop up until 18 months of age (Ottney, 1991). In the past, and still occasionally today, alcohol has been recommended to nursing mothers to aid in lactation. "Folklore relates that drinking small quantities of alcohol shortly before nursing increases milk yield, facilitates milk let-down, and relaxes both mother and infant. No scientific evidence supports any of these contentions, however" (Mennella & Beauchamp, 1991).

Alcohol passes freely into breast milk, nearly reaching maternal serum levels. Depending on the mother's alcohol intake breastfed babies are exposed to varying amounts of alcohol and effected accordingly. A recent study by Ives and Tepper (1990) reports that ethanol ingested during breast-feeding has a small, but statistically significant detrimental effect on motor, but not mental, development. Other studies have found that moderate amounts of alcohol consumed over a period of time during lactation may slow brain growth; large amounts consumed in one drinking episode may limit brain growth to an even greater extent (Lauwers & Woessner, 1990). Excessive amounts of alcohol have also been shown to interfere with the let-down response, limit parental effectiveness and result in serious conditions in the nursing baby (Lauwers & Woessner, 1990). Additionally, alcohol consumption has been demonstrated to reduce milk intake by infants (Mennella & Beauchamp, 1991). Some animal research has found that early and long-term exposure to

² The segregation of tissues into different organs in embryonic development (Miller & Keane, 1972).

flavours in the mother's milk may affect later flavour preferences, including that for alcohol—the implications of these findings for the human population has also raised some concern (Philips & Stainbrook, 1976 as cited in Mennella & Beauchamp, 1991). In summary, no safe level of alcohol consumption for breastfeeding women has been determined.

Studies which have examined critical periods for prenatal alcohol exposure suggest that cessation of drinking, even as late as the third trimester of pregnancy, can have benefits (Coles, 1994). For example, Coles et al. (1987) found that women who quit drinking during the second trimester have infants with measurable neurobehavioural deficits though these deficits were not as acute as in children whose mothers continued to drink for the duration of the pregnancy. Animal and human studies have also shown greater birthweights if mothers reduce their consumption during the third trimester (Ginsburg et al., 1991).

Authors generally concur that full-blown FAS is probably not due to a single binge episode, but is caused by regular consumption of alcohol throughout gestation. Continual exposure to alcohol during the entire period from conception to birth would account for the wide range of effects seen in the full-blown Fetal Alcohol Syndrome. Episodic binge drinking at high levels is viewed as resulting in partial expression of the syndrome, with the abnormalities being unique to the period of exposure (Secretary of Health..., 1990).

5.3 Metabolism of Alcohol by the Mother

In addition to the quantity of alcohol consumed during pregnancy and the gestational timing of alcohol exposure, the mother's ability

to metabolize alcohol influences the extent of damage to the fetus. The length of time the mother has been drinking prior to the pregnancy, her age, body weight, drinking style and the conditions of the pregnancy itself, all influence the mother's metabolism.

Women in advanced stages of alcoholism may have damaged livers which impair their ability to break down the alcohol, thus resulting in higher levels of alcohol in the fetal bloodstream (Ottney, 1991). Also, as a person ages, the body loses water, resulting in less dilution of the alcohol and in higher blood alcohol levels (Ottney, 1991). Pregnancy also typically raises blood alcohol levels to higher than usual non-pregnant levels (Ottney, 1991). An equivalent dose of alcohol will obviously have more of an effect on a woman of lower body weight as compared to a woman who weighs more. For a number of reasons, alcoholic women usually have lower than average body weights. Abel (1990) reports that mothers of FAS children, for whom weight data were available, were considerably below the average weight for height ranges. Finally, metabolism is also affected by the pattern of consumption—higher blood alcohol levels are reached during binge drinking when many drinks are consumed over a short period of time.

Some authors have suggested that women who are able to moderate their drinking in pregnancy are different in some essential way which, in turn, affects the outcome of their pregnancy. It may be that the severity of the drinking problem or stage of alcoholism is a more important factor than the duration or dose of alcohol consumed (Majewski, 1981 as cited in Little & Wendt, 1991).

5.4 Other Factors

One of the reasons it is difficult to conclusively attribute the cause of alcohol-related birth defects only to alcohol use is that there are many other prenatal and postnatal factors that may partially account for the results. The research literature is in agreement that prenatal alcohol intake is the primary cause of FAS. However, the research is much less clear on the relative role which other suspected risk factors play in contributing to, or worsening, alcohol-related birth defects. As stated by Ernhart (1991):

A concern of most investigators studying risk conditions in early child development are the correlations found between many other conditions of fetal, neonatal, and child development and the risk factors and outcomes of interest. While the problem is particularly troublesome for measures of psychological development, which are affected by numerous social and environmental circumstances, confounding [factors] can influence almost any outcome in epidemiological research.

Ernhart (1991) notes that most studies of fetal alcohol effects control for maternal smoking since smoking is often correlated with drinking, and has been found to be related to some outcomes such as lower birth weights. Ernhart also mentions maternal age, race, parental size, parental intelligence, quality of the child care environment, obstetric complications and gestational age as possible cofactors which researchers usually control for in their research. Similarly, Abel (1993) believes that women who use other drugs in addition to alcohol may be at higher risk for having a child with FAS.

In a paper devoted to reviewing the literature and developing a strategy for researching the effects of smoking and drinking during pregnancy on offspring learning disabilities,

Streissguth (1986) identifies several possibly confounding factors which may also influence the research findings. For example, at conception, genetic factors which may account for subsequent learning disabilities must be considered. To this end, Streissguth (1986) recommends that information be collected on maternal education (as a predictor of child IQ) and that a positive family history of learning disabilities should also be assessed. During gestation, it is also important to assess everything the mother ingests, particularly other drugs, nicotine, and types of food. As well, the health of the mother needs to be considered. It should be established if conditions such as diabetes, heart disease, exposure to viral and environmental toxins, as well as fevers, illnesses and infections occurred during the pregnancy. With respect to factors particular to the birth itself, delivery medications and gestational age should also be noted. Birth weight and the Apgar score³ may also be useful pieces of information in assessing certain outcomes. "Infancy is a time for environmental factors, such as diet, breast feeding and mother-infant interactions, to play an important role" (Streissguth, 1986, p. 35). In addition, serious illnesses and accidents during childhood must be considered. During the preschool years, Streissguth notes that preschool attendance, birth order and family density are possible interacting variables in studying learning disabilities. Streissguth (1986) notes that it is very important to attempt to assess and quantify the school and the quality of teaching, as these elements may account for learning disabilities.

Finally, and perhaps most importantly, many studies examining information on the background of children with alcohol-related birth defects suggest that there is a high degree of familial upheaval (Streissguth and Randels, 1988; Burd & Martsolf, 1989). Many children are

³ A method for determining an infant's condition at birth by scoring the heart rate, respiratory effort, muscle tone, reflex irritability and color (Miller & Keane, 1972).

never cared for by their biological mothers, many natural mothers die when the children are very young, and a high number live in multiple foster homes throughout their lives. Further research is needed to clarify the role which these poor postnatal environmental factors (e.g., multiple foster placements) may play in causing or exacerbating maladaptive behaviours.

Many research studies and literature reviews have been conducted which specifically examine the role of various other factors in affecting pregnancy outcome. The findings of these studies vary considerably depending on the factor(s) being studied as well as on the specific postnatal outcomes being evaluated. Due to this lack of consensus in the literature, it is impossible to concisely summarize the effects of certain factors such as smoking, nutrition, and child-rearing practices on postnatal outcome.

6. Can a Father Cause or Contribute to Alcohol-Related Birth Defects?

Paternal alcohol exposure and its contribution to alcohol-related birth defects has been the subject of much research recently. According to Abel (1992), this concern for the father's role has occurred for several reasons including: the knowledge that men generally drink more heavily than women, that many alcoholic women are married to alcoholic men, and that a majority of women with FAS children appear to have mates who are also alcoholic. Abel (1992) believes that the effects of maternal drinking are very likely confounded by paternal drinking. In light of emerging studies that are beginning to link fathers' exposure to various toxins (e.g., lead, some anti-cancer medications, nuclear radiation and dioxin-containing herbicides) to birth defects (Purvis, 1990), it does not seem unreasonable to suggest that paternal consumption of alcohol, a known teratogen, has some effects as well.

One frequently quoted study by Little and Sing (1987) (as cited in Abel, 1992) documented decreased birthweight associated with heavy paternal alcohol consumption during the month of conception. Several recent animal studies have investigated the influence of paternal alcohol consumption on offspring under well-controlled conditions (Cicero, 1994). These studies have shown that in animals there are effects on offspring which result exclusively from paternal alcohol exposure, though the mechanisms by which these deficits occur are not clear. Some of the interesting findings that have emerged from these studies include:

- in pre-pubescent male rats exposure to alcohol adversely affected puberty and sexual maturation, with reproductive functioning returning to normal within two to three weeks following alcohol withdrawal;
- despite the restoration of normal hormonal functioning following a drug-free period at the time of sexual maturation, future offspring of these animals still exhibited abnormal development; this suggests that there may be long-term, possibly permanent consequences of heavy alcohol use by human male adolescents on the development of the offspring they bear later as adults;
- deficits found in the offspring of male animals exposed to alcohol were gender-specific. Male offspring had significant disturbances in reproductive hormonal functioning (e.g., lower levels of testosterone) as well as a deficit in spatial learning abilities. In comparison, female offspring had abnormal levels of stress-related hormones causing them to respond differently to stress than those offspring whose fathers were not exposed to alcohol heavily prior to conception. Additionally, in these rats the deficits in offspring were selective (only hormones that influence reproduction (in males) and stress (in females) were affected, while the only behavioural deficit noted was spatial learning abilities in male offspring. Cicero (1994) states that: "...these results are highly consistent with the observations in humans, in that offspring of alcohol fathers, as opposed to offspring [of alcohol mothers] suffering from FAS, are not grossly malformed or impaired but have pronounced selective intellectual and functional deficits."

It is interesting to note that, because of these recent findings showing that heavy paternal alcohol consumption may have long-lasting, possibly permanent effects causing deficits in future offspring, some researchers are beginning to question the findings of earlier genetic research which has assumed that some genetic trait predisposes offspring of male alcoholics to alcoholism. "Few investigators have considered the possibility that these deficits could be due

to alcohol's being a direct gonadal toxicant or teratogenic agent" (Cicero, 1994, p. 40). This possibility will likely be an important issue for future research into alcoholism.

The father's role in prevention of alcohol-related birth defects is often discussed in a general way in the FAS literature. It is felt that the father can play a significant role by acting as a role model in reducing consumption and by encouraging and supporting the woman to reduce her alcohol intake (Ottney, 1991; Canada. Standing Committee..., 1992; Addiction Research Foundation, 1992).

7. What Is the Incidence of FAS and Other Alcohol-Related Birth Defects?

Due to differences in how researchers consider sample populations and sub-groups (such as Aboriginal populations) in calculating incidence rates in the general population, many different incidence rate estimates are found in the literature. The reliability and accuracy of diagnosis can also influence resulting incidence rates (Cordero et al., 1994). For example, the application of the standard diagnostic criteria for FAS across cultures is seen as problematic and is believed to account for some of the higher FAS incidence rates among Natives (Bray & Anderson, 1989). Streissguth and LaDue (1987) are quoted in a report produced by the Yukon Government in 1991 as saying that “the prevalence of FAS is a function of the drinking habits of women of childbearing ages in the population and the skill and experience of the local diagnosticians” (Yukon. Government, 1991). Additionally, incidence rates are influenced by the accuracy and thoroughness of the methods used for recording and reporting FAS (Cordero et al., 1994).

In the literature, researchers express caution about the fact that, while Fetal Alcohol Syndrome does occur more frequently in some population groups, it definitely is not exclusive to any racial or ethnic group (Burgess & Streissguth, 1992). The possible reasons for high rates of FAS within certain ethnic groups are many—some researchers point to possible genetic or ethnic/racial risk factors (Abel, 1990 as cited in Canada. Standing Committee...,

1992). However, there is no conclusive evidence as yet linking any of these risk factors to the expression of FAS in particular ethnic sub-populations. Many authors believe that drinking patterns and norms vary substantially by community and subgroup in society and affect the prevalence of FAS (May, 1991; Abel, 1993).

Similarly, a link between socioeconomic status and incidence of FAS has not been scientifically established. Streissguth and LaDue (1985) found evidence of an interaction of socioeconomic status with the relationship between maternal drinking and fetal alcohol effects, though this finding has not been consistently repeated in the literature.

There is general agreement that most estimates are likely very conservative as they reflect only those individuals who have actually been identified, referred and diagnosed (Burgess & Streissguth, 1992). As discussed previously in the section entitled “Difficulties Diagnosing FAS,” the problems associated with making a diagnosis of FAS, particularly in the newborn, likely result in only the most severe cases being diagnosed and included in the estimates. Also, FAS incidence rates do not include those children who die shortly after birth.

It is infinitely more difficult to identify children who do not have FAS, but who may have other alcohol-related birth defects (Abel, 1993). In some of the research literature, incidence rates of fetal alcohol effects are estimated at two to three times that for FAS.

The following section briefly presents estimates of the incidence of FAS in North America and Canada.

7.1 Incidence in North America

The most frequently cited estimate of FAS incidence among the general population in the Western world is .33 per 1000 (or one per 3000) live births (Abel & Sokol, 1991). This estimate was compiled from several studies which prospectively collected data of consecutive pregnancies. Abel and Sokol (1991) found incidence rates ranged from 0 to 1.58/1000 depending on the ethnic and socioeconomic status of the population being studied. On average, they found the incidence rate for Caucasians to be .29/1000, while the incidence rate for Afro-Americans was .48/1000. An estimate for North American Aborigines was not included in their overall estimates as data were not available. However, due to the relatively small numbers of Native American Aborigines in the general population, they did not think that the overall FAS incidence rate would be greatly affected.

Originally, Abel and Sokol estimated the incidence of FAS at 1.9 per 1000 live births. Their "revised conservative estimate of the incidence of FAS" (i.e., .33/1000 live births), made in 1991 was considerably lower for two major reasons (Abel & Sokol, 1991): (1) only prospective study data were analyzed (as opposed to mostly retrospective studies that were used in 1987), and (2) racial susceptibilities for FAS or ethnic population differences were taken into consideration when projecting prevalence rates to the general population. Based on Abel and Sokol's (1987) original estimate for the incidence of FAS, it was concluded that mental retardation as a result of FAS occurred at approximately the same rate as Down's Syndrome in the United States (Ives, 1987). It should be noted that several articles written after 1991 still quote Abel and Sokol's (1987) original, higher FAS incidence rate of

1.9/1000 (Robinson et al., 1992; Canada. Standing Committee..., 1992).

In the United States, prevention of FAS is a national health priority and is included in the "Healthy People 2000" objectives for health promotion and disease prevention. The U.S. Department of Health and Human Services has specified that they want to reduce the rate of FAS to no more than 1.2 cases per 10,000 live births by the year 2000 (as well, they want to increase abstinence from alcohol consumption by pregnant women by 20%) (U.S. DHHS, 1995). In order to evaluate the effectiveness of their prevention efforts, good surveillance techniques are required and continue to be refined. Baseline data for their objective has been obtained from two sources: (1) the National Birth Defects Monitoring Program of the Centers for Disease Control and Prevention, and (2) the Metropolitan Atlanta Congenital Defects Program.

In 1993, data from the National Birth Defects Monitoring Program (BDMP), which examines hospital discharge data for newborns, pegged FAS as occurring at a rate of 6.7 per 10,000 newborns. The 1993 rate was more than six times higher than the rate reported in 1979. Possible reasons for this increase include that the incidence of FAS may be increasing, or that there has been an increase in awareness and diagnosis of FAS by primary care physicians. It should be noted that the medical discharge code used to determine the presence of FAS (i.e., as defined by the International Classification of Diseases, Ninth Revision, Clinical Modification, code 760.71) is not specific to FAS and may also include a broad range of adverse effects attributed to maternal alcohol consumption during pregnancy. For this reason, the estimate for FAS may be an overestimate (U.S. DHHS, 1995).

The Metropolitan Atlanta Congenital Defects Program monitors all births occurring in the metropolitan Atlanta area. Unlike the BDMP, it uses multiple sources to identify diagnoses of FAS in newborns and throughout the first year of life. In 1992, they reported a rate of 3.3 cases of FAS per 10,000 live births.

Cordero and associates (1994) discuss the importance of having a good national FAS surveillance program. Ultimately, epidemiologic data are needed to evaluate changes in the rate of FAS over time, so that the success of FAS prevention activities can be monitored. They state that: "The first task in improving surveillance is to develop a uniform case definition of FAS that optimally balances sensitivity with precision and can be used for surveillance" (p. 85). This may include the development of sensitive screening instruments. The second task, they state, is to ensure uniformity and compliance in methods of reporting and receiving the data across the country.

7.2 Incidence in Canada

The Fifth Report of the Standing Committee on Health and Welfare, Social Affairs, Seniors and the Status of Women (Canada. Standing Committee..., 1992) states that there is no known incidence rate of FAS in Canada primarily due to diagnostic difficulties. Based on incidence rates found in the United States, Health and Welfare Canada estimates, seemingly without knowledge of Abel and Sokol's (1987) revised estimate, that there are one to two per 1000 children born with FAS. They believe this is a low estimate as it does not take into account high rates among Aboriginal Canadians and possibly other socially disadvantaged groups.

Studies conducted in British Columbia and Manitoba highlight the wide range of estimates

of incidence rates of FAS and FAS/FAE. A study conducted in an isolated northern Native community in British Columbia found that 14 of 123 children had FAS while eight had fetal alcohol effects (Robinson et al., 1987). This translated to an unexpectedly high combined incidence rate for FAS and fetal alcohol effects of 190/1000 in the community. Two-thirds of the 22 children with FAS or fetal alcohol effects were mentally retarded. In Manitoba, a study done in 1990 at a teaching hospital in Winnipeg assessed the minimum incidence of FAS at five per 1,000 live births (Single et al., 1994).

The exact rate of occurrence of FAS in Alberta is also unknown. In Alberta, during 1994/95, 96 discharges released from hospital were identified to Alberta Health as having FAS. Of these, 17 had a primary diagnosis of FAS (Alberta Health, 1996). Given the difficulties around diagnosing and inconsistent reporting, this number may underestimate the actual number of people with FAS in this province.

7.3 FAS as a Cause of Mental Retardation

In 1987, Abel and Sokol published an article which stated that "mental retardation is a cardinal feature of FAS and is now recognized as the leading known cause of mental retardation in the Western world" (Abel & Sokol, 1987, p. 51). This much-quoted statement was based on the recognition that mental retardation is one of the more serious features of Fetal Alcohol Syndrome, and was based on Abel and Sokol's initial (i.e., 1987) FAS prevalence rate estimate of 1.9/1000 live births. Other authors disagree that FAS is the leading known cause of mental retardation and have placed it as the third leading cause, behind Down's Syndrome and neural tube defects (Ottney, 1991).

Given Abel and Sokol's more recent (1991) lower estimate of FAS incidence (.3/1000), it seems reasonable to conclude, as some authors have, that Fetal Alcohol Syndrome is currently the leading cause of preventable birth defects and is one of the three leading known causes of mental retardation. In certain populations where the incidence of FAS is high, it will be the leading cause of mental retardation. It is important to note that only about 10% of the causes of all cases of mental retardation can currently be identified (Ottney, 1991).

8. Is There a Concern About Labelling Children as Having Alcohol-Related Birth Defects?

In the literature, early diagnosis of FAS and identification of individuals suspected of having alcohol-related birth defects is generally recommended so that their progress can be monitored and appropriate medical, educational and social interventions can be made (Streissguth & Giunta, 1988). LaDue (1993) recommends the early identification of children at risk for the following reasons:

- it is important for implementing needed services;
- it helps maximize the potential outcome for the child;
- it is important for identifying women and families at risk; and
- it helps in providing needed services for families at risk and maximizing the chances for the family to remain intact.

Several authors caution against making a diagnosis of FAS until all other possible causes have been considered and eliminated. FAS is similar to several other patterns of malformations and conditions including: Cornelia deLange Syndrome (Yukon. Government, 1991; Clarren & Smith, 1978), Noonan Syndrome (Clarren & Smith, 1978; Streissguth et al., 1991), DiGeorge Syndrome (Abel, 1990), infants born to mothers with Phenylketonuria and infants of epileptic mothers who take Dilantin (anticonvulsant medication)

(Ernhart, 1991), as well as infants with Blume Syndrome, Dubowitz Syndrome or chromosomal problems (Streissguth et al., 1991).

Obtaining an accurate assessment of the mother's alcohol consumption during pregnancy before labelling a child as having FAS is also often stressed as "misdiagnosis of FAS can burden parents with a lifetime of unnecessary guilt" (Ottney, 1991, p. 24).

The utility of labelling a child in the school setting as having an alcohol-related birth defect is debated in the literature. In a book which discusses strategies for managing children who have been prenatally exposed to drugs and alcohol, it is stated:

In general, labels which indicate a child has experienced prenatal drug or alcohol exposure will not be useful in the school or community setting. Labels describing learning styles ("kinesthetic learner"), behavioral attributes ("energetic and active"), or social capabilities ("shy and quiet") can be more practical because they suggest steps a teacher or other provider can take to reinforce strengths or address weaknesses. Diagnostic labels that describe specific attention disorders ("attention deficit disorder with hyperactivity") or learning problems ("difficulties with visual organization") likewise prescribe actions that will help the child. "Drug-exposed" is a term which refers to a medical condition. It can inform a physician of specific problems to look for and important assessments to make. It has no similar utility for the teacher or provider outside the health arena, especially those who work with school-age children. In school settings, in fact, such a label is more likely to handicap a child unfairly (Villareal et al., 1991, pp. 129-130).

Abel (1993, p. 18) agrees, stating "when he gets to school, he's labelled as Fetal Alcohol Effects or Alcohol-Related Birth Defects. Then the teacher mainlines him some place as a trouble-maker, instead of doing something for these

kids. Once you label someone, that label stays with them."

On the other hand, some researchers believe that knowledge that a child has FAS is critical as it enables appropriate interventions to be developed to meet the special needs which usually accompany FAS. Giunta and Streissguth (1988, p. 456) state: "Teachers at all levels should have training and information about fetal alcohol syndrome in order to help them recognize and understand the problems facing these children. Familiarity with FAS can help teachers set realistic performance expectations and tailor their teaching methods to the special needs of students with FAS." In another article that provides guiding principles for educational programs designed for students who are prenatally exposed to alcohol, it is stated: "to maximize the effectiveness of educational programs, it is crucial to begin early to guide the learning of appropriate, functional skills and to decrease the occurrence of inappropriate behaviors" (Burgess & Streissguth, 1992, p. 26). Burgess and Streissguth also point out that a correct diagnosis can be a relief as it helps to explain academic and social difficulties that the children encounter in school. However, they note the concern about the social and emotional repercussions which can result from labelling and caution that it "is a step that must be taken carefully and thoughtfully" (Burgess & Streissguth, 1992, p. 25).

Much of the recent discussion in the scientific literature regarding labelling revolves around the issue of the practical value of a label. For example, in a discussion about abandoning the term fetal alcohol effects, Aase and associates (1995) express concern that decisions about qualifying for special funding or educational services should be based on demonstrated patient need as opposed to arbi-

trary categories of diagnosis (i.e., the label of fetal alcohol effects). "The results of specific physical, psychological, and behavioral testing form the best basis for such decisions, and pediatricians armed with this information can be effective advocates for their patients in helping to obtain appropriate services as well as helping change the present inadequate qualification categories" (Aase, et al., 1995, p. 429).

9. What Are Some Intervention Strategies for Those Affected by Alcohol-Related Birth Defects?

To date, there has been a lack of research on effective intervention strategies for those affected by FAS and other ARBDs (Weiner & Morse, 1994). Much of the information on intervention strategies comes from parent reports (from newsletters and support groups) and a few books based on parents' and teachers' experiences in working with children who have FAS. With this in mind, this section provides an overview of the needs of individuals with FAS along with some general intervention strategies for parents and others caring for individuals with FAS as well as for educators.

9.1 Needs of Children With FAS

Before adequate intervention strategies can be designed, the challenges of raising and educating children with FAS must be understood. Most children with FAS have mild to moderate behaviour problems and IQs ranging from 45 to 110 (Weiner & Morse, 1994). "Each child with FAS is unique, exhibiting some but not all of the behavioral characteristics; the children are alike, however, in that they all show some learning difficulties and behavior problems" (Burgess & Streissguth, 1992 as cited in Weiner & Morse, 1994, p. 68). According to Weiner and Morse (1994), the literature has focused more on the children most critically affected by FAS (e.g., those with IQs below 50 and severe behavioural problems); thus, predicting nega-

tive outcomes for all children with FAS. They argue that such pessimistic predictions do not consider the enormous diversity of this population and the potential benefits of targeted intervention strategies.

It is important to recognize the individual and specific needs of people with FAS, "who often have multidisciplinary problems that involve the home, the schools, the health care system, vocational training, the criminal justice system, and the community" (Streissguth, 1994, p. 79). A person with FAS may need a variety of services to address their health, family, and educational needs.

Children with FAS typically have multiple health care needs. Initially, the diagnosis of FAS needs to be made by a specialist (e.g., geneticist) or trained professional who understands FAS and knows what to look for and what questions to ask (Davis, 1992). An early diagnosis of FAS is the important first step in linking children with appropriate intervention strategies during the most formative years (Weiner & Morse, 1994). Children with FAS may require treatment for any congenital anomalies. For instance, malformations of the ear, eye, heart defects, and cleft lip and cleft palate that can accompany FAS may need ongoing medical treatment. As children with FAS become adolescents and adults, additional areas of medical concern include birth control and sex education (Giunta & Streissguth, 1988).

Giunta and Streissguth (1988) also identify family needs of children with FAS. Some research suggests that children with FAS are at a higher than average risk for physical abuse, sexual abuse, and neglect. Such children are often living in high-risk environments where the biological mother is struggling with sobriety and has few resources and support. As a result, children with FAS need close monitoring.

Giunta and Streissguth (1988) suggest that a loving, structured family setting with clear guidelines and clear communication is ideal. When foster or adoptive placement is required, people who are calm and low-key, secure with themselves, and who live stable and predictable lives have the highest success (Giunta & Streissguth, 1988). Unfortunately, the difficulties faced by parents of children with FAS (e.g., difficulties with the children themselves and with finding support services) often results in high turnover in foster placements (Weiner & Morse, 1994). Parents need adequate access to information about FAS from a variety of sources including helping professionals and support groups. Informed parents are better able to realistically assess their children's behavioural and learning abilities and to help teachers, health care providers, and other caregivers understand and provide for their children's needs (Weiner & Morse, 1994).

A third area to consider for children with FAS is educational needs. As infants, some severely affected FAS children can benefit from early mental and motor stimulation provided by infant-stimulation programs. Developmental delays in language skills and hyperactivity are sometimes first noted in preschool years. It is beneficial for preschool teachers to be aware of and able to recognize deficits in these areas so that they can make appropriate referrals for diagnosis and testing (Giunta & Streissguth, 1988).

Giunta and Streissguth (1988) suggest that teachers at all levels should have information and training about FAS to help recognize and understand the problems facing these children. Otherwise, some behaviours may be interpreted incorrectly compromising appropriate interventions (Weiner & Morse, 1994). To illustrate, a neurobehavioural issue, such as aggression

or temper tantrums, may result in some children with FAS being inappropriately labeled as bad or malicious. Teachers can benefit from guidelines and protocols to address the unique problems of children with FAS. For example, because of short attention spans and distractibility, some children with FAS may function better in a small classroom setting with a clear and consistent routine. Many children with FAS plateau academically by high school. At this stage, Giunta and Streissguth (1988) recommend that education for adolescents with FAS provide some attention to basic life skills (e.g., money management, communication, safety, etc.) to foster some degree of independence.

9.2 General Intervention Strategies

The goal of intervention strategies is to give all those affected by FAS every opportunity to thrive in society and to reach their maximum potential (Weiner & Morse, 1994). In the past, what were thought to be suitable environmental interventions and educational opportunities for populations of mentally retarded children (e.g., those with Down's Syndrome) have often not been effective for those with FAS. In particular, this has been the case for people with FAS who are not mentally retarded and for those with mild cognitive deficits that are compounded by attentional deficits (Streissguth, 1994).

Even though there has not been systematic studies of effective strategies for FAS, parents and teachers, independently have developed intervention strategies that have had some positive outcomes in children with FAS. Weiner and Morse (1994) organize the FAS intervention strategies into three categories: (1) environment; (2) learning skills; and (3) consistency. These strategies mainly focus on central ner-

vous system deficits that affect intelligence and behaviour.

Environment - "Restructuring the child's environment is the primary technique consistently described by parents, teachers, and others who have observed children with FAS" (Weiner & Morse, 1994, p. 68). The purpose of this strategy is to remove barriers to the child's progress. For example, a teacher might arrange a classroom by setting up specific work and play areas. As well, because children with FAS are prone to hypersensitivity, some suggest keeping work areas clear and free from distraction and putting away any materials not currently in use (Tanner-Halverson, 1993 as cited in Weiner & Morse, 1994; Davis, 1992). Another example is planning teaching activities so as not to overstimulate—ensuring reading occurs in a quiet room, for instance.

Learning skills - Teaching children how to learn is another intervention strategy. Children affected by FAS seem to learn and use information differently than the average child (Kodituwakku et al., 1992 as cited in Weiner & Morse, 1994). One teacher in a northern rural community uses several techniques such as role playing and peer tutoring to help students with FAS learn (Winick, 1993). Role playing can be effective in teaching these children real-life related lessons—how to understand consequences and appropriate behaviour, for example. Another teacher emphasizes the importance of adults mediating the learning experience to show children with FAS learning skills such as how to keep themselves on a task and to improve their memory (Phillpot & Harrison, 1993).

Consistency - A third intervention strategy is consistency in the environment, including the behaviour and responses of people in the child's world (Weiner & Morse, 1994). This

strategy may be applied in school settings and in the child's home. For example, one parent found that the most successful school programs for her son with FAS included: (1) teachers who were explicit and consistent when addressing the children; (2) visual aids and classroom arrangement that reinforced class rules and activities; and (3) program routine that varied little from day to day (Caldwell, 1993). Davis (1992) also states that children with FAS function best when there is structure, order, and routine. These children benefit from a learning environment that is consistent and structured and recognizes their individual learning and emotional needs (Villarreal et al., 1991).

9.3 Guidelines for Parents and Others Caring for Individuals With ARBDs

In a follow-up study of 61 adolescents and adults previously diagnosed with FAS or fetal alcohol effects, Streissguth and associates (1988) gathered data from a variety of sources including the people caring for patients with FAS and fetal alcohol effects (e.g., foster and adoptive parents). Based on the interviews of parents, they suggest the following guidelines for caregivers.

Information - Parents need to learn all they can about the signs and symptoms of FAS as well as the medical, social, and behavioural consequences. This information is important for people planning on becoming foster or adoptive parents and for recovering alcohol dependent mothers. It is important for parents to understand that the child has damage to the brain that causes behavioural and developmental disturbances. The information will assist in setting realistic goals and expectations for their children and themselves and help prepare

them for the difficult task of coordinating health and social services for their children.

Behaviour Management - Parents of young children with FAS may need support and guidance in behavioural management of hyperactivity and behavioural disturbances. Understanding the relationship between difficult early experiences (e.g., abuse or neglect) and a child's disturbed behaviour in a later different home environment can help foster or adoptive parents deal realistically and supportively with the variety of symptoms that can occur (e.g., withdrawal, unusual fear).

Advocate - Parents play an important role in assuring appropriate school placement of the children with FAS. They understand their children's needs best and can be effective advocates for their children at school and in the community.

Adolescents' needs - Parents of an adolescent with FAS must deal with several tasks including: sexual maturity in a developmentally disabled person; the adolescent's decreasing satisfaction at school and plateauing academic skills and subsequent need for vocational skills; and managing the adolescent's leisure time, interpersonal relations, and independence.

Respite - Parents of children, adolescents, or adults with FAS need respite care for themselves. Raising children with FAS can be demanding and tiring. It is important that parents arrange time away on a regular basis. Rest is crucial to a parent's ability to keep doing a good job. Getting rest will involve substitute child care which could be arranged through a social worker. Parent support groups and/or counselling in dealing with the stresses of raising children with FAS may be necessary for parents.

Long-term planning - Parents need to consider long-term planning for financial and guardianship issues. The assistance of a social worker is often needed in arranging for available subsidies and in accessing appropriate community resources and services.

9.4 Guidelines for Educators of Children Affected by ARBD

In addition to guidelines for parents, Streissguth also outlines four guiding principles for educational programs of students who have been prenatally exposed to alcohol or drugs (Burgess & Streissguth, 1992).

Early intervention - Alcohol-related birth defects are lifelong disabilities. To maximize the effectiveness of education programs, it is important to begin early to guide learning of appropriate, functional skills and to decrease the occurrence of inappropriate behaviours. Because FAS is a medical diagnosis, a diagnosis cannot be made by educators. Instead, guidelines need to be in place to ensure students suspected of having FAS or other alcohol-related birth defects receive appropriate assessment, diagnosis, and treatment.

Targeting functional skills - Students with FAS and fetal alcohol effects need academic skills, but they need to be complemented by the skills that will help students live and succeed in the world. They argue that the key is to focus on both the present and future environments in which students will live and work and to teach skills specific to those settings. Consistent with Burgess and Streissguth (1992), Davis (1992) also contends that as students with FAS and fetal alcohol effects get older, they need less emphasis on academics and more on basic living skills, social skills, anger management and vocational training.

Teaching communication skills - Even though communication skills among students with FAS and fetal alcohol effects vary, there appears to be a general discrepancy between their verbal language and their ability to actually communicate effectively. Educators need to recognize students' attempts to communicate and to direct those attempts into appropriate words and behaviours.

Teaching social skills - Social isolation can be a problem among young people with FAS and FAE because of their poor communication and inability to predict consequences of their behaviour and impulsivity. It is important to teach appropriate social skills whenever opportunities present themselves, whether it be in the hallway, cafeteria, or classroom.

10. What Are Some Strategies to Prevent Alcohol-Related Birth Defects?

This section provides a cursory overview of prevention strategies in the area of alcohol-related birth defects (ARBDs). Although an in-depth review of prevention programming would be valuable, such a task is beyond the scope of this report.

Over the past few years, prevention of ARBDs has received more attention in the literature. In particular, the literature discusses various types of prevention strategies, such as warning labels on alcoholic beverages and prenatal programs screening for women at risk for having children with alcohol-related birth defects. This section highlights typical prevention strategies, prevention goals and messages, target groups, common prevention approaches, barriers to women entering addictions treatment, and the effectiveness of prevention efforts.

10.1 Prevention Strategies

Typical prevention strategies have been information-based, relying on pamphlets, posters, television media campaigns, warning labels on alcoholic beverages, prenatal education and counselling, training for professionals such as health care providers, and community-based programs (Carr, 1994; Hankin, 1994). It is generally agreed that comprehensive programs incorporating multiple strategies and implemented at the community level are the most effective for preventing FAS (U.S. NIAAA, 1987;

Blume, 1992). Prevention programs vary in the number and type strategies used. Some of the more common prevention strategies are highlighted below.

Public Education

Public education includes various strategies to convey information on ARBDs, including:

- public awareness campaigns;
- age-appropriate education in elementary and secondary schools (ASAM, 1992);
- public education through addictions agencies (ASAM, 1992);
- print materials: for example, posters and specialty items (e.g., calenders, placemats);
- warning signs at point of sale of alcoholic beverages (ASAM, 1992; Canada. Government, 1992; Addiction Research Foundation, 1992); and
- warning labels on alcoholic beverages (ASAM, 1992; Canada. Government, 1992; Addiction Research Foundation, 1992).

Warning labels, in particular, have generated much discussion. The labeling of alcoholic beverages is seen as a tool for increasing the awareness of the general public that alcohol consumption during pregnancy can be harmful to the fetus (Canada, Health and..., 1992). However, there is considerable debate about the effectiveness of warning labels. Some research suggests that warning labels only have a small effect (Abel, 1993; Holder, 1994). For example, one study examining the introduction of warning labels in the U.S. found that the warning labels on alcohol beverages were being noticed, read and recalled by some consumers in general (Kaskutas & Greenfield, 1992). However, there was no change in the already high knowledge level of the health risks included on the warning labels and there were little differences in behavioural intentions between

those who saw and those who did not see the labels. Similarly, a study of pregnant women in Detroit found that alcoholic beverage warning labels resulted in a small decrease in drinking among women who were non-risk drinkers, but did not influence women drinking at risky levels (Hankin, 1994).

There are two sides to the warning label debate. Proponents of warning labels argue that the government has an obligation to inform the public of an established health danger. The public has the right to know of risks associated with a product. In contrast, opponents focus more on the impact of warning labels. They contend that there is no evidence that warning labels will alter drinking behaviour among those women most at risk for having children with Fetal Alcohol Syndrome (Abel, 1993). Information alone cannot make problem drinkers stop (Blume, 1992). Moreover, warning labels can be misleading and create unnecessary fear or public concern, especially for those who are light drinkers. For example, the Motherisk program in Toronto has found high levels of anxiety in some pregnant women who equate mild drinking with high risk for FAS; these women are ready to terminate an otherwise wanted pregnancy (Sharp, 1995). In addition, there is concern that the public is being bombarded by so many health warnings on products that the warnings are being viewed with indifference and often ignored.

Both sides seem to agree that warning labels are one of many ways for increasing awareness, but that as a singular prevention strategy, they are ineffective. However, those against warning labels argue that if you are going to spend limited resources, it is important to target the resources on the people who are most at risk for FAS—women with alcohol problems (Abel, 1993).

Professional Education

Another important strategy in preventing ARBDs is the education and mobilization of professionals. Professional education typically includes education or training of allied professionals dealing with women, children, and families affected by ARBDs such as FAS. For example, health care professionals working with pregnant women and young children need a broad base of knowledge and skills so they are able to identify a high-risk pregnancy and adverse pregnancy outcomes (Bondy et al., 1993). Some suggested prevention strategies targeting professionals include:

- improved education aimed at early diagnosis of addictions problems in women in health care settings (e.g., revise curriculum for all health professionals' training programs to include knowledge of alcohol and effects, skills, and tools);
- dissemination of information to public health nurses, social service workers, day care workers, educators, correctional workers, and addictions counsellors about ARBDs and related support and resources available;
- in-service training for physicians and nurses; and
- a 'train the trainer' approach to educating professionals about ARBDs.

Some research suggests that a key element in women's decisions to reduce or stop drinking during pregnancy is the advice given by their physicians and other health care providers (Huyter, 1993). Yet, health care professionals are often reluctant to inquire about alcohol use, therefore reducing the extent to which ARBDs could be preventable. For example, a study of New Jersey physicians showed that they do not routinely ask their pregnant patients about alcohol consumption for many reasons, including physician bias due to their own drinking, lack of training, poor awareness of the problem and its effects, time limitations,

fear of offending patients, and the belief that patients will not tell the truth about their drinking (Donovan, 1991). Similarly, a Canadian study of Saskatchewan physicians found family physicians and general practitioners who graduated before 1974 were less likely than recent graduates to be aware of FAS and ask their patients about alcohol use during pregnancy (Nanson et al., 1995). However, these physicians were more likely to feel comfortable discussing alcohol-related issues. Overall, this study found Saskatchewan physicians are aware of FAS, but they expressed a need for more information on FAS for parents as well as physician training materials and information about where to refer patients with FAS and parents with alcohol-related problems.

There is a need to create a system that responds quickly to women with substance abuse problems. The ultimate goal is to eliminate the traditional barriers that exist between the provision of health care and the treatment and prevention of substance abuse, including reducing the negative attitudes among professionals with regard to pregnant substance abusers (U.S. DHHS, 1995).

Treatment Services

Educational efforts to increase awareness of ARBDs are not enough to prevent all occurrences of alcohol-related birth defects. Another way to approach ARBD prevention is to focus prevention efforts on the people who are most at risk for having a child with FAS. Abel (1993) refers to focused prevention as identifying women who are having problems, and then targeting one's resources to these women to increase the number of normal pregnancies. Usually, this process involves screening pregnant women when they seek prenatal care and outreach efforts to contact women not likely to enter the traditional health care system.

Early identification of problem drinkers -

In recent years, a variety of studies have demonstrated the feasibility of screening for heavy drinking and/or alcohol problems in women within the health care system (Blume, 1992). Early identification of a drinking problem is difficult because of the tendency of people who drink to deny or underestimate their drinking. The two most commonly cited brief questionnaires used to identify women who are at risk for heavy drinking are the T-ACE and the TWEAK (Blume, 1992; Abel, 1993; Russel, 1994).

Based on research of two earlier developed alcoholism screening instruments, the CAGE and Michigan Alcoholism Screening Test (MAST), Dr. Sokol and his colleagues at Wayne State University in Detroit developed the T-ACE questionnaire (Blume, 1992; Russel, 1994). As shown in Table 10.1, it includes four questions; a score of two or more indicates high-risk drinking.

The TWEAK questionnaire was developed by Dr. Russel at the Research Institute on Addictions in Buffalo, New York. It combines questions from the MAST, CAGE, and T-ACE tests that have been found most effective in identifying women drinkers. Also shown in Table 10.1, it includes five questions and, like the T-ACE, a total score of two or more indicates high-risk drinking.

Table 10.1 Screening Instruments

The T-ACE Questionnaire

1. **T** How many drinks does it take to make you feel high?
(TOLERANCE - more than two drinks scores two points)
2. **A** Have people ANNOYED you by criticizing your drinking?
(a positive response scores one point)
3. **C** Have you felt you ought to CUT DOWN on your drinking?
(a positive response scores one point)
4. **E** Have you ever had a drink first thing in the morning to steady your nerves or get rid of a hangover?
(EYE-OPENER - a positive response scores one point)

The TWEAK Questionnaire

1. **T** How many drinks can you hold?
(TOLERANCE - if a woman reports holding more than five drinks without falling asleep or passing out she scores two points)
 2. **W** Have close friends or relatives WORRIED or complained about your drinking in the past year?
(a positive response scores two points)
 3. **E** Do you sometimes take a drink in the morning when you first get up?
(EYE-OPENER - a positive response scores one point)
 4. **A** Has a friend or family member ever told you about things you said or did while you were drinking that you could not remember?
(AMNESIA - a positive response scores one point)
 5. **K(C)** Do you sometimes feel the need to CUT DOWN on your drinking?
(a positive response scores one point)
-

For women identified as risk drinkers, a thorough assessment may be indicated. For many women, accurate information about the risk involved with and advice about drinking during pregnancy are sufficient to eliminate hazardous drinking (Rosett et al., 1983 as cited in Russel, 1994). However, follow-up and referral for addictions treatment are necessary for pregnant women who drink heavily and are unable to reduce their alcohol intake (Russel, 1994; ASAM, 1992). It is important to have effective links between health care and addictions treatment services.

Outreach services - It is often difficult to attract women with substance abuse problems into treatment. Combined fear of reprisal from significant others or the community, even legal repercussions, dissuade these women from seeking treatment. Outreach services help to get women to services they would not otherwise receive, and it encourages them to use available health care and social services (U.S. DHHS, 1995). Employing outreach workers who are indigenous to the neighbourhoods and the culture tends to facilitate such women seeking treatment.

In the late 1980s, a Pregnancy Outreach Program (POP) was implemented in British Columbia (Asante & Robinson, 1990; Radford, 1992). The purpose of the program was to improve the reproductive health of women at risk. The outreach service was located apart from traditional health and hospital settings—at friendship centres and child development centres, for example. The program consisted of four components including: group sessions to encourage peer support/education; milk and vitamin supplements for clients in financial need; individual counselling and support, usually through outreach by lay counsellors; and referral to other community agencies.

Other treatment services - Treatment services not only include treatment services for problem-drinking pregnant women, but also includes women with substance use problems in general (U.S. NIAAA, 1987). This means accessible addiction treatment services designed to meet women's needs, plus patient and family education in primary care settings, obstetrical practices, and in addictions treatment programs are other important prevention strategies (ASAM, 1992).

10.2 Prevention Goals and Messages

Generally, prevention programs for ARBDs are aimed at reducing the number of alcohol-related birth defects (U.S. NIAAA, 1987; Hankin, 1994). In keeping with this overall goal, other specific goals reflecting various prevention strategies include the following (U.S. NIAAA, 1987):

- to increase the awareness of the general public about the hazards associated with alcohol during pregnancy;
- to encourage women of childbearing age to avoid alcohol and unnecessary drugs during pregnancy;
- to encourage women who are drinking during pregnancy to reduce their consumption to increase their chances of having healthy babies in the current pregnancy and subsequent pregnancies;
- to urge women with drinking problems to seek and accept treatment;
- to influence health, social service, and education professionals to provide education on alcohol and drug effects to all patients, clients, and students prior to and during pregnancy; and
- to identify and link affected families and children with resources offering emotional support, treatment, and rehabilitation.

Within the context of these goals, prevention programs convey different messages depending on the target group. One common message to pregnant women concerns the safe level of alcohol consumption during pregnancy. Given the inconsistent research findings on a safe level of alcohol consumption during pregnancy, most prevention programs advise abstinence from alcohol for pregnant women (ASAM, 1992; Canada. Government, 1992). To a lesser extent, some programs also recommend abstinence from alcohol for women planning pregnancy and for nursing mothers (ASAM, 1992). A slightly more moderate position is taken by the Addiction Research Foundation (1992, p. 4) which recommends that physicians advise pregnant women about alcohol consumption, emphasizing that when it comes to pregnancy, "less is better and none is best."

10.3 Target Groups

Since the late 1970s, prevention efforts have focused heavily on educational strategies targeting various groups (Smith & Coles, 1991):

- pregnant women, especially pregnant women who are heavy drinkers and considered high-risk (U.S. NIAAA, 1987);
- women of childbearing age, including female adolescents (U.S. NIAAA, 1987); the education of all women is important regardless of the extent of their drinking, to permit women the opportunity to make informed decisions about alcohol consumption prior to conception and during pregnancy;
- health professionals (e.g., family physicians, obstetricians, prenatal care professionals, and pediatricians etc.) to improve identification and treatment of pregnant drinkers as well as children with FAS (U.S. NIAAA, 1987); and
- the general public, including parents, grandparents, and friends who may play a role in influencing the pregnant women. As well, school age

children are the future parents and need to be aware of the risks for FAS.

There has been some discussion about the extent to which prevention strategies should be aimed at general populations versus targeting women (and their families) at risk for having children with alcohol-related birth defects. Some, such as Dr. Streissguth, believe that a combination of general population and targeted strategies are needed (Bondy et al., 1993) and others advocate more focused prevention on women at risk (Abel, 1993). Most delegates at an FAS workshop in Halifax felt that education messages aimed at the general public were important to maintain high levels of awareness and help to establish community norms that discourage drinking during pregnancy (Bondy et al., 1993). As well, they recognized that targeted prevention efforts cannot be ignored. A woman who has had one child with FAS requires intensive intervention aimed at preventing another adverse pregnancy. Agreeing with the need to target at risk women, Abel reasons that if you are going to spend limited resources, it is important to target them (Abel, 1993). He argues that focusing prevention on at risk women is more effective in increasing the number of normal pregnancies than general broad prevention efforts such as warning labels on alcoholic beverages.

Acknowledging that ARBDs are not solely the responsibility of women, some also suggest that information about the risks of ARBDs should be targeted at all men and women of child-bearing age (Canada. Government, 1992). Men who understand the risk are more likely to support their partners in efforts to reduce drinking or abstain from alcohol during pregnancy.

10.4 Prevention Approaches

Over the past few years, there has been more discussion in the literature on underlying approaches or frameworks of ARBD prevention efforts. Of the various approaches found, the two most common were the health promotion approach (Loock, 1992; Loney et al., 1994; Ouellet, 1988), and the public health approach (U.S. DHHS, 1993; May, 1995).

Health Promotion Approach

A health promotion approach helps to broaden our understanding of ARBDs. More specifically, health promotion: (1) “recognizes the broad range of determinants of health; (2) it emphasizes the context of individuals’ lifestyles, rather than viewing behaviour as simply a personal choice; (3) it acknowledges the importance of addressing social inequities in health; and (4) it calls for community action and coordinated, intersectoral policies to address health issues” (Loney et al., 1994, p. 248). This broad approach recognizes that alcohol use occurs in a social context. Although FAS is a distinct medical syndrome, the main cause (excessive alcohol consumption during pregnancy) is embedded in everyday patterns of living that are reinforced by social, economic and cultural factors (Loney et al., 1994). Within this approach, some health promotion strategies for preventing ARBDs include (Loock, 1992):

- fostering public participation (e.g., server intervention programs, community-based action groups);
- strengthening community health services (e.g., strong links among hospital, community health, pregnancy outreach, and addictions treatment services);
- coordinating healthy public policy (e.g., comprehensive school health curriculum, legislation

involving labeling, lifestyle advertising, and alcohol accessibility); and

- coordinating policies between sectors (e.g., inter-governmental committees with representation from all relevant departments, such as health, justice, social services, education, and alcohol and drugs).

These strategies highlight the interdisciplinary nature of ARBDs and point out that prevention efforts can be integrated with other related health promotion efforts. For example, substance abuse prevention can be incorporated as part of broader health promotion goals such as increasing participation in community health prenatal programs to promote healthier women and healthier babies (U.S. DHHS, 1995).

At a program level, Ouellet (1988) proposes four general principles be followed when implementing a program using a health promotion approach.

- Begin where people are ‘at.’ Initiate programs first in areas that people are familiar (e.g., schools, community groups) and initiate policies and programs at the level of common understanding and commitment. Within this context, health promoting behaviour changes during pregnancy are more likely to lead to lasting healthy lifestyles.
- Involve the community. This means identifying and understanding the needs and characteristics of each community.
- Make the most of your resources. In times of restraint, it is important to identify available resources and look for creative and diverse ways of using existing expertise and facilities.
- Adopt a comprehensive approach involving a mix of strategies that support and enhance each other. For instance, strategies might include: a mass media component to increase awareness; an information/education component to ensure informed decision-making; and environmental measures to reduce barriers and enable healthy choices.

Public Health Approach

Within the health care field, prevention efforts are commonly divided into three levels of prevention: primary, secondary, and tertiary prevention (U.S. DHHS, 1993). In terms of ARBDs, primary prevention aims to avoid or prevent the problem of FAS before it exists; in other words, the goal is to have no children born with FAS or other ARBDs (May, 1995; U.S. DHHS, 1993). The most common strategy used in primary prevention is public education (e.g., public awareness campaigns and education in schools) to inform the population about the hazards of substance use during pregnancy.

Secondary prevention typically seeks to reduce the duration of a now existing problem in some individuals (May, 1995). In relation to FAS, secondary prevention usually focuses on identifying and intervening with persons at risk for, or beginning to experience, substance use problems through prenatal clinics or outreach in areas of high risk environments (U.S. DHHS, 1993). The main goal is to reduce alcohol and other drug intake during pregnancy.

Tertiary prevention "refers to rehabilitation after disease has occurred" (U.S. DHHS, 1993, p. 10). In terms of ARBDs, this usually means treating substance use problems or treatment and management of complications, impairments, and disabilities caused by alcohol and other drug consumption during pregnancy (U.S. DHHS, 1993; May, 1995). With FAS, tertiary prevention typically focuses on preventing a woman who has given birth to one child with FAS from having another child with FAS or other ARBDs. As well, tertiary prevention efforts aim to reduce the physical, emotional and social problems of the child with ARBDs (Bondy et al., 1993; May, 1995).

Philip May (1995), Professor and Director at the University of New Mexico Center on Addictions, combines what has been learned from the addictions literature about alcohol and women with the prevention levels (i.e., primary, secondary, tertiary) of the public health approach (U.S. DHHS, 1993). The goal of this comprehensive approach is to reduce the number of children being born with ARBDs by applying the appropriate level of prevention according to the level of maternal alcohol consumption. May explains that "the levels of prevention are mutually complementary in that primary prevention can be applied to all members of the populations, but secondary and tertiary level preventions are reserved for those women who are heavier drinkers and therefore at greater current risk for producing children with ARBD or FAS" (May, 1995, p. 1565).

The prevention levels work in conjunction and, when implemented together, will have a maximum impact. Primary prevention efforts are directed at abstainers and light alcohol users to reinforce their level of alcohol consumption. Secondary prevention efforts (e.g., screening, brief counselling) are directed at persons with moderate consumption and tertiary prevention efforts (e.g., addiction treatment services) are aimed at those with substantial or heavy alcohol consumption (U.S. DHHS, 1993).

This approach clearly ties ARBD prevention within the broader context of the alcohol consumption and emphasizes the importance of a comprehensive, coordinated system to prevent ARBDs. Also, this approach underscores that the vast majority of the population are abstainers or light drinkers who will respond to primary prevention measures. Secondary and tertiary prevention are only appropriate for a small proportion of the population. According

to May (1995), studies show that about 25% of all women in prenatal clinics continue to drink, but most women reduce their drinking during pregnancy, and heavy drinking women are a small subset of this group of women.

Common Characteristics of Approaches

The various approaches or models discussed in the literature to prevent ARBDs do not fit easily into a single approach or model. The issues are complex and solutions must be developed cooperatively, encompassing the viewpoints of many professionals and multiple disciplines or service sectors (U.S. DHHS, 1993). A review of approaches by the U.S. Center for Substance Abuse Prevention found several common themes across the various approaches (U.S. DHHS, 1993). Some of these common characteristics are as follows:

- prevention must be comprehensive—a range of services along a continuum of care is needed from before pregnancy to long after pregnancy;
- prevention should include primary, secondary and tertiary prevention efforts—primary prevention should not be the exclusive focus; secondary and tertiary prevention are important to prevent another generation of problems;
- prevention must be collaborative—cooperation and collaboration among many social agencies and community groups allows for a comprehensive range of services responsive to women and their families' needs;
- prevention must be community-based—the most effective prevention strategies actively involve the communities for whom the services are intended;
- prevention must be culturally appropriate—an understanding of communities' beliefs and values, knowledge and attitudes is important in order to effectively communicate health information in culturally appropriate ways; and

- prevention must be nonjudgemental and non-punitive—judgemental and punitive interventions are counterproductive to women seeking services.

10.5 Barriers to Women Entering Addictions Treatment

Barriers to ARBD prevention programs are most commonly discussed in relation to intervention or treatment of pregnant women with substance use problems or, more generally, in relation to women seeking treatment for addictions (Smith & Coles, 1991; MacPhee, 1992).

According to Smith and Coles (1991), female drug users as a group are less likely to enter and complete treatment than their male counterparts (Reed, 1988, & Vannicelli, 1984, as cited in Smith & Coles, 1991). Pregnant women may be even less likely to seek treatment for several reasons (Smith & Coles, 1991):

- the tremendous stigma attached to being pregnant and addicted;
- less support of family and friends for entering and completing treatment; and
- failure on the part of the treatment system to acknowledge women's needs (e.g., some programs will not accept pregnant women fearing increased liability, few programs include provisions for childcare).

Similar types of barriers were cited by MacPhee (1992) at an FAS symposium in Vancouver. She outlined several barriers to high-risk women seeking treatment based on her experience at an emergency short-term day care and women's drop-in in Vancouver (Crabtree Corner YWCA):

- no protocols or prioritizing services for pregnant women in detoxification or treatment centres;
- no information available on FAS;

- no women-only detoxification areas (many of these women have been sexually abused and they do not want to use services that include men);
- no area alcohol and drug services;
- no prenatal and postnatal programs;
- no safe, affordable housing for pregnant women with alcohol and drug problems;
- no community-based services for pregnant women to provide coordinated and comprehensive care;
- no support for birth parents of children with FAS;
- a lack of sensitivity to the cultural and literacy needs of these women; and
- fear by pregnant women who are using alcohol and drugs that, if they seek treatment, their newborn baby will be apprehended.

In the U.S., an added barrier is the recent legislative trends toward more punitive treatment of alcohol or drug abusing pregnant women (Smith & Coles, 1991). Some women have been placed in jail, attempts have been made to curb or terminate custody rights, and some pregnant drug users have been prosecuted for acts of harm against the fetus (Farr, 1995). In the past few years, at least seven U.S. states have either attempted unsuccessfully to or are in the process of enacting fetal abuse laws.

In Canada, legal action is not a common practice. However, there was one case in the Yukon in 1985 where a territorial judge used provisions under the Children's Act and granted an application ordering a pregnant woman to undergo alcohol counselling. Yukon government officials stress the provision is only used in extreme circumstances, when usual measures such as counselling and intervention by family and community workers have failed to

convince a woman to stop drinking while pregnant (Radford, 1995).

There is no evidence that this legal approach is either socially effective or economically sound (Farr, 1995). Some suggest that criminalization of the pregnant substance abuser minimizes the impact of addiction and places the behaviour within the context of morality (Smith & Coles, 1991). This approach is counterproductive in that the likelihood of such women seeking prenatal care and other services is reduced due to fear of arrest or possible imprisonment. As well, such an approach creates additional problems. For instance, sending mothers to prison keeps them from parenting and requires placement alternatives for their drug-exposed infants.

10.6 Effectiveness of Program Efforts

To evaluate changes in the rate of ARBDs over time, research is needed on the incidence and prevalence of FAS and other ARBDs in the population (e.g., among certain groups such as Aboriginal Canadians). In addition, it is important that prevention efforts are evaluated to determine their effect on women's knowledge and behaviour, especially among heavy-drinking women (Sokol & Abel, 1992).

Unfortunately, there is little documented research on the effectiveness of specific prevention programs or strategies (Schorling, 1993). Some evidence does suggest that public education strategies, such as mass media campaigns, have been effective in increasing awareness and changing drinking behaviour of pregnant women who are social drinkers (Smith & Coles, 1991). However, there is little evidence that such prevention efforts bring about behaviour change in the women who are

heavy drinkers and therefore at greatest risk (Smith & Coles, 1991; Sokol & Abel, 1992).

Similarly, in a review of studies evaluating prenatal education and counselling programs, Schorling (1993) found a significant proportion of women continued to drink throughout pregnancy regardless of the counselling programs. However, the majority of women reduced or eliminated alcohol consumption by the end of their pregnancies. This reduction in drinking was also found in the control groups suggesting that other factors outside the prenatal counselling programs were likely related to the reduction in drinking.

There has been some evaluations of the effectiveness of professional education programs on FAS (Blume, 1992; Hankin, 1994). Most of the evaluations reported an increase in the number of physicians asking their patients about their alcohol consumption, but over time this change was less marked illustrating the need for ongoing training, encouragement, and support to make professional education effective (Blume, 1992).

11. Conclusion

ARBDs is a very complex and emotional issue. Despite the abundant research in the area, several gaps in knowledge and much uncertainty remain. It is not possible to provide straightforward answers to most questions that are commonly asked about alcohol consumption during pregnancy. Some issues that emerge in the literature as being particularly contentious are:

- the terminology in the area is ambiguous;
- the diagnosis and identification of children with alcohol-related birth defects is very difficult—because of this, children could be labelled incorrectly and caution in making a diagnosis is urged;
- the research findings do not support any safe level or pattern of alcohol consumption during pregnancy; and
- there is inadequate information on the incidence of ARBDs such as FAS—differing estimates are found throughout the literature, and none are available for Canada.

From the literature, it is apparent that ARBDs can be addressed on many different levels—from public education efforts to increase awareness of ARBDs to comprehensive, multidisciplinary prevention efforts involving several programs and agencies. Because of the tremendous cost and ramifications of ARBDs to individuals with ARBDs, parents and other caregivers, educators, social service professionals, and health care professionals, a balanced and multidisciplinary response to this difficult and complex problem is needed.

11. References

- Aase, J.M. (1994). Clinical recognition of FAS: Difficulties of detection and diagnosis. *Alcohol Health & Research World (Special Focus: Alcohol-Related Birth Defects)*, 18(1), 5-9.
- Aase, J.M., Jones, K.L. & Clarren, S.K. (1995). Do we need the term "FAE"? *Pediatrics*, 95(3), 428-430.
- Abel, E.L. (1990). *Fetal Alcohol Syndrome*. Oradell, New Jersey: Medical Economics Books.
- Abel, E.L. (1992). Paternal exposure to alcohol. In T.B. Sonderegger (Ed.), *Prenatal Substance Abuse: Research Findings and Clinical Implications* (pp. 132-160). Baltimore: The John Hopkins University Press.
- Abel, E.L. (1993, July 27). *Fetal Alcohol Syndrome: Impact on the individual and society*. Presentation of BACCHUS Canada at the University of Toronto, Toronto, Ontario.
- Abel, E.L. & Sokol, R.J. (1987). Incidence of Fetal Alcohol Syndrome and economic impact of FAS-related anomalies. *Drug and Alcohol Dependence*, 19, 51-70.
- Abel, E.L., & Sokol, R.J. (1991). A revised conservative estimate of the incidence of FAS and its economic impact. *Alcoholism: Clinical and Experimental Research*, 15(3), 514-524.
- Addiction Research Foundation. (1992, August). Substance use and reproduction: Risks, rights and responsibilities. *Best Advice*. Toronto, Author.
- Alberta Health (February, 1996). *Fetal Alcohol Syndrome: Hospital morbidity data base - 1994-95* (response prepared by Regional Services of Alberta Health to an AADAC information request).
- American Society of Addiction Medicine (ASAM). (1992). Public policy statement on the use of alcohol and other drugs during pregnancy. *Journal of Addictive Diseases*, 11(4), 125-127.
- Asante, K.O. & Robinson, G.C. (1990). Pregnancy outreach program in British Columbia: The prevention of alcohol-related birth defects. *Canadian Journal of Public Health*, 81(1), 76-77.
- Best, L.C. (1993). *Guiding Our Children Beyond Risk: A Handbook for Caretakers of Prenatally Drug Exposed Children (2nd ed.)*. San Francisco, California: The Clearinghouse for Drug Exposed Children. Division of Behavioural and Development Pediatrics.
- Blume, S.B. (1992). Chapter 5: Preventing FAS/FAE-the pregnant woman. In *What You Can Do to Prevent Fetal Alcohol Syndrome: A Professional's Guide* (pp. 35-41). Minneapolis: Johnson Institute.
- Bondy, S., Muscovitch, F.A., & Rankin, J.G. (1993, October). Summary of eight key issues: Research workshop on priorities, problems and strategies. In the workshop report *Prevention of Adverse Fetal Effects of Ethanol in Canada: A Research Workshop on Priorities, Problems and Strategies*, Halifax, Nova Scotia.
- Bray, D.L., & Anderson, P.D. (1989). Appraisal of the epidemiology of Fetal Alcohol Syndrome among Canadian Native peoples. *Canadian Journal of Public Health*, 80, 42-45.

- Burd, L. & Martsof, J.T. (1989). Fetal Alcohol Syndrome: Diagnosis and syndromal variability. *Physiology and Behavior*, 46(1), 39-43.
- Burgess, D.M. & Streissguth, A.P. (1992, September). FAS and FAE: Principles for educators. *Phi Delta Kappa*, 74(1), 24-29.
- Caldwell, S. (1993). Nurturing the delicate rose. In J. Kleinfeld and S. Wescott (Eds.), *Fantastic Antone Succeeds! Experiences in Education Children with Fetal Alcohol Syndrome* (pp. 97-129). Fairbanks, AK: University of Alaska Press.
- Canada. Government. (1992, December). *Fetal Alcohol Syndrome: 84 From Awareness to Prevention. Government response to the fifth report of the Standing Committee of the House of Commons on Health and Welfare, Social Affairs, Seniors and the Status of Women*. Ottawa: Author.
- Canada. Health and Welfare (1992). *Report on the Symposium on Fetal Alcohol Syndrome and Fetal Alcohol Effects*. Ottawa: Author.
- Canada. Standing Committee on Health and Welfare, Social Affairs, Seniors and the Status of Women. (1992, June). *Fetal Alcohol Syndrome: A Preventable Tragedy*. Ottawa: Author
- Canadian Centre on Substance Abuse (CCSA) National Working Group on Policy (1994). *Fetal Alcohol Syndrome: An Issue of Child and Family Health*. (A policy discussion paper). Ontario: Canadian Centre on Substance Abuse.
- Carr, R.A. (1994). *Field-Test of a Peer Educator Workbook for the Prevention of Fetal Alcohol Syndrome and Fetal Alcohol Effects*. Victoria, B.C.: Peer Resources.
- Casiro, O. (1992). The effects of alcohol consumption during pregnancy. *Alcoholism Foundation of Manitoba Directions*, 1(3), 1-3.
- Cicero, T.J. (1994). Effects of paternal exposure to alcohol on offspring development. *Alcohol Health & Research World (Special Focus: alcohol-related birth defects)*, 18(1), 37-41.
- Clark, S. (1993). *Alcohol Consumption & Fetal Abnormalities (Fetal Alcohol Syndrome)*. Edmonton, Alberta: Paper prepared for the Alberta Alcohol and Drug Abuse Commission.
- Clarren, S.K. & Smith, D.W. (1978). The Fetal Alcohol Syndrome. *The New England Journal of Medicine*, 298(19), 1063-1067.
- Cordero, J.F., Floyd, R.L., Martin, M.L., Davis, M., & Hymbaugh, K. (1994). Tracking the prevalence of FAS. *Alcohol Health & Research World (Special Focus: alcohol-related birth defects)*, 18(1), 82-85.
- Coles, C. (1994). Critical periods for prenatal alcohol exposure: Evidence from animal and human studies. *Alcohol Health & Research World (Special Focus: alcohol-related birth defects)*, 18(1), 22-29.
- Coles, C.D., Brown, R.T., Smith, I.E., Platzman, K.A., Erickson, S., & Falek, A. (1991). Effects of prenatal alcohol exposure at school age. I. Physical and cognitive development. *Neurotoxicology and Teratology*, 13, 357-367.
- Coles, C.D., Smith, I.E., & Falek, A. (1987). Prenatal alcohol exposure and infant behavior: Immediate effects and implications for later development. *Advances in Alcohol and Substance Abuse*, 6(4), 87-104.
- Conry, J. (1990). Neuropsychological deficits in Fetal Alcohol Syndrome and fetal alcohol effects. *Alcoholism: Clinical and Experimental Research*, 14(5), 650-655.

- Davis, D. (1992). *Reaching Out*. Seattle, Washington: Author.
- Day, N.L., Goldschmidt, L., Robles, N., Richardson, G., Cornelius, M., Taylor, P., Geva, D., & Stoffer, D. (1991). Prenatal alcohol exposure and offspring growth at 18 months of age: The predictive validity of two measures of drinking. *Alcoholism: Clinical and Experimental Research*, *15*(6), 914-918.
- Day, N.L. & Richardson, G.A. (1991). Prenatal alcohol exposure: A continuum of effects. *Seminars in Perinatology*, *15*(4), 271-279.
- Day, N.L., Robles, N., Richardson, G., Geva, D., Taylor, P., Scher, M., Stoffer, D., Cornelius, M., & Goldschmidt, L. (1991). The effects of prenatal alcohol use on the growth of children at three years of age. *Alcoholism: Clinical and Experimental Research*, *15*(1), 67-71.
- D'Entremont, D.M. (1990). *Intervention strategies for school age children with Fetal Alcohol Syndrome*. Paper written at the University of Southern Maine.
- Donovan, C.L. (1991). Factors predisposing, enabling and reinforcing routine screening of patients for preventing Fetal Alcohol Syndrome: A survey of New Jersey physicians. *Journal of Drug Education*, *21*(1), 35-42.
- Ernhart, C.B. (1991). Clinical correlations between ethanol intake and Fetal Alcohol Syndrome. In M. Galanter (Ed.), *Recent Developments in Alcoholism: Volume 9 Children of Alcoholics* (pp. 127-150). New York: Plenum Press.
- Ernhart, C.B., Sokol, R.J., Ager, J.W., Morrow-Tlucak, M., & Martier, S. (1992, June). Alcohol-related birth defects: Assessing the risk. *Annals of the New York Academy of Sciences*, *562*, 159-172.
- Farr, K.A. (1995). Fetal abuse and the criminalization of behavior during pregnancy. *Crime and Delinquency*, *41*(2), 235-245.
- Giberson, P.K. & Weinberg, J. (1992). Fetal Alcohol Syndrome and functioning of the immune system. *Alcohol Health and Research World*, *16*(1), 29-38.
- Ginsburg, K.A., Blacker, C.M., Abel, E.L., & Sokol, R.J. (1991). Fetal alcohol exposure and adverse pregnancy outcomes. *Contributions to Gynecology and Obstetrics*, *18*, 115-129.
- Giunta, C.T., & Streissguth, A.P. (1988, September). Patients with Fetal Alcohol Syndrome and their caretakers. *Social Casework: The Journal of Contemporary Social Work*, 453-459.
- Ives, E.J. (1987, December). The Fetal Alcohol Syndrome. *MEDICINE: North America*, 3074-3080.
- Ives, T.J. & Tepper, R.S. (1990). Drug use in pregnancy and lactation. *Pharmacotherapeutics for the Primary Care Physician*, *17*(3), 623-645.
- Hankin, J.R. (1994). FAS prevention strategies: Passive and active measures. *Alcohol Health and Research World (Special Focus: Alcohol-Related Birth Defects)*, *18*(1), 62-66.
- Holder, H.D. (1994). Public health approaches to the reduction of alcohol problems. *Substance Abuse*, *15*(2), 123-138.
- Huyter, E.A. (1993, November). Fetal Alcohol Syndrome and fetal alcohol effects. A report in the workshop *Drinking and Pregnancy: The Risks* (pp. 1-11). (Halifax, Nova Scotia). Nova Scotia: Department of Health, Drug Dependency Services Division.

- Jacobson, J.L. & Jacobson, S.W. (1994). Prenatal alcohol exposure and neurobehavioral development: Where is the threshold? *Alcohol Health & Research World (Special Focus: alcohol-related birth defects)*, 18(1), 30-36.
- Jones, K.L. (1986). Fetal Alcohol Syndrome. *Pediatrics in Review*, 8(4), 122-126.
- Jones, K.L., & Smith, D.W. (1973, November 3). Recognition of the Fetal Alcohol Syndrome in early infancy. *The Lancet*, 999-1001.
- Kaskutas, L. & Greenfield, T.K. (1992). First effects of warning labels on alcoholic beverage containers. *Drug and Alcohol Dependence*, 31(1), 1-14.
- Kearns, B. (1979, June). *Fetal Alcohol Syndrome*. Paper presented at the Canadian Public Health Association 70th Conference, Winnipeg, Manitoba.
- Kleinfeld, J. & Wescott, S. (Eds.) (1993). *Fantastic Antone Succeeds! Experiences in Educating Children with Fetal Alcohol Syndrome*. Fairbanks, Alaska: The University of Alaska Press.
- LaDue, R.A. (1993). *Psychosocial needs associated with Fetal Alcohol Syndrome: Practical guidelines for parents and caretakers*. Paper presented at conference on Fetal Alcohol Syndrome and Fetal Alcohol Effect: A Community Perspective, Lethbridge, Alberta.
- LaDue, R.A., Streissguth, A.P., & Randels, S.P. (1992). Clinical considerations pertaining to adolescents and adults with Fetal Alcohol Syndrome. In T.B. Sonderegger (Ed.), *Prenatal Substance Abuse: Research Findings and Clinical Implications* (pp. 104-131). Baltimore: The John Hopkins University Press.
- Landesman-Dwyer, S., Ragozin, A.S. & Little, R.E. (1981). Behavioral correlates of prenatal alcohol exposure: A four-year follow-up study. *Neurobehavioral Toxicology and Teratology*, 3, 187-193.
- Lauwers, J. & Woessner, C. (1990). *Counseling the nursing mother: A reference handbook for health care providers and lay counselors* (2nd ed.). New York: Avery Publishing Group Inc.
- Little, R.E. & Wendt, J.K. (1991). The effects of maternal drinking in the reproductive period: An epidemiologic review. *Journal of Substance Abuse*, 3, 187-204.
- Loney, E.A., Green, K.L., & Nanson, J.L. (1994). A health promotion perspective on the House of Commons report "Fetal Alcohol Syndrome: A preventable tragedy." *Canadian Journal of Public Health*, 85(4), 248-251.
- Loock, C.A. (1992, October 2). Fetal Alcohol Syndrome and fetal alcohol effects: Common, expensive, and preventable. *Report on the Symposium on Fetal Alcohol Syndrome and Fetal Alcohol Effects* (pp. 14-19). (Vancouver, British Columbia) Ottawa: Health and Welfare Canada.
- MacPhee, B. (1992, October 2). Day two plenary: Approaches that are working—Panel discussion. *Report on the Symposium on Fetal Alcohol Syndrome and Fetal Alcohol Effects* (pp. 64-68). (Vancouver, British Columbia) Ottawa: Health and Welfare Canada.
- May, P.A. (1991). Fetal alcohol effects among North American Indians. *Alcohol Health & Research World*, 15(3), 239-248.

- May, P.A. (1995). A multiple-level, comprehensive approach to the prevention of Fetal Alcohol Syndrome (FAS) and other alcohol-related birth defects (ARBDs). *The International Journal of the Addictions*, 30(12), 1549-1602.
- Mennella, J.A. & Beauchamp, G.K. (1991). The transfer of alcohol to human milk: Effects on flavor and the infant's behavior. *New England Journal of Medicine*, 325, 981-985.
- Miller, B.F. & Keane, C.B. (1972). *Encyclopedia and Dictionary of Medicine and Nursing*. Toronto: W.B. Saunders.
- Morse, B. (1993). Information processing. In J. Kleinfeld and S. Wescott (Eds.), *Fantastic Antone Succeeds! Experiences in Education Children with Fetal Alcohol Syndrome* (pp. 23-36). Fairbanks, AK: University of Alaska Press.
- Nanson, J. L., Bolaria, R., Snyder, R.E., Morse, B.A., & Weiner, L. (1995). Physician awareness of Fetal Alcohol Syndrome: A survey of pediatricians and general practitioners. *Canadian Medical Association Journal*, 152(7), 1071-1076.
- Olson, H.C., Sampson, P.D., Barr, H., Streissguth, A.P., & Bookstein, F.L. (1992). Prenatal exposure to alcohol and school problems in late childhood: A longitudinal prospective study. *Development and Psychopathology*, 4(3), 341-359.
- Ottney, J.R. (1991). *Fetal Alcohol Syndrome: Facts and Choices. A Guide for the teacher*. Madison, Wisconsin: University of Wisconsin.
- Ouellet, B. (1988). Changing perspectives on the role of health promotion: Implications of Fetal Alcohol Syndrome. In G.C. Robinson & R.W. Armstrong (Eds.), *Alcohol and Child/Family Health* (pp. 3-17). Vancouver, B.C.: University of British Columbia.
- Overholser, J.C. (1990). Fetal Alcohol Syndrome: A review of the disorder. *Journal of Contemporary Psychotherapy*, 20(3), 163-176.
- Phillipson, R. (1988). The Fetal Alcohol Syndrome—Recent international statistics. *Australia and New Zealand Journal of Developmental Disabilities*, 14(3&4), 211-217.
- Phillpot, B. & Harrison, N. (1993). A one-room schoolhouse for children with FAS/FAE. In J. Kleinfeld and S. Wescott (Eds.), *Fantastic Antone Succeeds! Experiences in Education Children with Fetal Alcohol Syndrome* (pp. 233-250). Fairbanks, AK: University of Alaska Press.
- Purvis, A. (1990, November 26). The sins of the fathers. *Time*, p. 68.
- Radford, T. (1992, December). British Columbia. *Action News*. 111(6), 1-3.
- Radford, T. (1995). Yukon's treatment plan for pregnant women causes stir. *Action News*. V1(2), 5.
- Riley, E.P., Mattson, S.N., Sowell, E.R., Jernigan, T.L., Sobel, D.F., & Jones, K.L. (1995). Abnormalities of the corpus callosum in children prenatally exposed to alcohol. *Alcoholism: Clinical and Experimental Research*, 19(5), 1198-1202.
- Robinson, G.C., Armstrong, R.W., Moczuk, I.B., & Loock, C.A. (1992). Knowledge of fetal alcohol syndrome among native Indians. *Canadian Journal of Public Health*, 83(5), 337-338.
- Robinson, G.C., Conry, J.L., & Conry, R.F. (1987). Clinical profile and prevalence of Fetal Alcohol Syndrome in an isolated community in British Columbia. *Canadian Medical Association Journal*, 137, 203-207.

- Roman, E., Beral, V., & Zuckerman, B. (1988). The relation between alcohol consumption and pregnancy outcome in humans: A critique. In H. Kalter (Ed.), *Issues and Reviews in Teratology* (Vol. 4, pp. 205-235). New York: Plenum Press.
- Russell, M. (1991, May-June). Clinical implications of recent research on the Fetal Alcohol Syndrome. *Bulletin of the New York Academy of Medicine*, 67(3), 207-222.
- Russell, M. (1994). New assessment tools for risk drinking during pregnancy. *Alcohol Health and Research World (Special Focus: Alcohol-Related Birth Defects)*, 18(1), 55-61.
- Schorling, J.B. (1993). The prevention of prenatal alcohol use: A critical analysis of intervention studies. *Journal of Studies on Alcohol*, 54(3), 261-267.
- Secretary of Health & Human Services (1990). *Seventh Special Report to the U.S. Congress on Alcohol & Health* (DHHS Publication No. ADM 90-1656). Washington, D.C.: U.S. Government Printing Office.
- Single, E., Ashley, M.J., Riley, D., & McKenzie, D. (1992, February 27). *Canadian Centre on Substance Abuse: Statement on Fetal Alcohol Effects (FAE)*, 1-10.
- Single, E., MacLennan, A., & MacNeil, P. (1994). *Horizons 1994: Alcohol and Other Drug Use in Canada*. Ontario: Health Canada and the Canadian Centre on Substance Abuse.
- Sharp, A. (1995, September/October). FAS-status quo in Canada. *Healthy Drinking*, 48-49.
- Smith, I.E., & Coles, C.D. (1991). Multilevel intervention for prevention of Fetal Alcohol Syndrome and effects of prenatal alcohol exposure. In M. Galanter (Ed.), *Recent Developments in Alcoholism: Volume 9 Children of Alcoholics* (pp. 165-180). New York: Plenum Press.
- Sokol, R.J. (1981). Alcohol and abnormal outcomes of pregnancy. *Canadian Medical Association Journal*, 125, 143-148.
- Sokol, R.J. & Abel, E.L. (1992). Risk factors for alcohol-related birth defects: Threshold, susceptibility, and prevention. In T.B. Sonderegger (Ed.), *Prenatal Substance Abuse: Research Findings and Clinical Implications* (pp. 90-103). Baltimore: The John Hopkins University Press.
- Sokol, R.J. & Clarren, S.K. (1989). Guidelines for use of terminology describing the impact of prenatal alcohol on the offspring. *Alcoholism: Clinical and Experimental Research*, 13(4), 597-598.
- Streissguth, A.P. (1986). Smoking and drinking during pregnancy and offspring learning disabilities: A review of the literature and development of a research strategy. In M. Lewis (Ed.), *Learning Disabilities and Prenatal Risk* (pp. 28-67). Chicago: University of Illinois Press.
- Streissguth, A.P. (1988). Long term effects of Fetal Alcohol Syndrome. In G.C. Robinson & R.W. Armstrong (Eds.), *Alcohol and Child/Family Health* (pp. 135-151). Vancouver, B.C.: University of British Columbia.
- Streissguth, A.P. (1991). Neonatal effects of alcohol use. In J. Kinney (Ed.), *Clinical Manual of Substance Abuse* (pp. 202-204). Toronto: Mosby Year Book.

- Streissguth, A.P. (1994). A long-term perspective of FAS. *Alcohol Health & Research World (Special Focus: Alcohol-Related Birth Defects)*, 18(1), 74-81.
- Streissguth, A.P., Clarren, S.K., Randels, S.P., LaDue, R.A., Aase, J.M., & Smith, D.F. (1991). Fetal Alcohol Syndrome in adolescents and adults. *Journal of the American Medical Association*, 266(8), 1077.
- Streissguth, A.P., and Giunta, C.T. (1988). Mental health and health needs of infants and preschool children with Fetal Alcohol Syndrome. *International Journal of Family Psychiatry*, 9, 29-47.
- Streissguth, A.P., and LaDue, R.A. (1985). Psychological and behavioural effects in children prenatally exposed to alcohol. *Alcohol Health and Research World*, 10(1), 6-12.
- Streissguth, A.P., LaDue, R.A., & Randels, S.P. (1988). *A Manual on Adolescents and Adults with Fetal Alcohol Syndrome with Special Reference to American Indians* (2nd ed.). Seattle, Washington: University of Washington.
- Streissguth, A.P., & Randels, S. (1988). Long term effects of Fetal Alcohol Syndrome. In G.C. Robinson & R.W. Armstrong (Eds.), *Alcohol and Child/Family Health* (pp. 116-130). Vancouver, B.C.: University of British Columbia.
- Streissguth, A.P., Sampson, P.D., & Barr, H.M. (1989). Neurobehavioral dose-response effects of prenatal alcohol exposure in humans from infancy to adulthood. *Annals New York Academy of Sciences, Part V. Alcohol*, 145-158.
- U.S. Department of Health and Human Services (DHHS) (1995). Preventing and treating drug abuse in pregnant and parenting women. In B.J. Seitz de Martinez (Ed.) *Teaching Children Affected by Prenatal Drug Exposure* (pp. 239-252). Bloomington, Indiana: Phi Delta Kappa.
- U.S. Department of Health and Human Services (DHHS) (1993). *Center for Substance Abuse Prevention Technical Report 9: Toward Preventing Perinatal Abuse of Alcohol, Tobacco, and Other Drugs*. Rockville, M.D.: author.
- U.S. National Institute on Alcohol Abuse and Alcoholism (NIAAA). (1991, July). Fetal Alcohol Syndrome. *Alcohol Alert*, (13), 1-3.
- U.S. National Institution on Alcohol Abuse and Alcoholism (NIAAA). (1987). *Program Strategies for Preventing Fetal Alcohol Syndrome and Alcohol-Related Birth Defects*. (DHHS Publication No. ADM 87-1482). Washington, D.C.: U.S. Government Printing Office.
- Villarreal, S.F., McKinney, L. & Quackenbush, M. (1991). *Handle with Care: Helping Children Prenatally Exposed to Drugs and Alcohol*. Santa Cruz, CA: ETR Associates.
- Weiner, L. & Morse, B.A. (1994). Intervention and the child with FAS. *Alcohol Health & Research World (Special Focus: Alcohol-Related Birth Defects)*, 18(1), 67-72.
- Winick, P. (1993). Mainstreaming children with FAS in a small rural school. In J. Kleinfeld and S. Wescott (Eds.), *Fantastic Antone Succeeds! Experiences in Education Children with Fetal Alcohol Syndrome* (pp. 223-231). Fairbanks, AK: University of Alaska Press.
- Yukon. Government. (1991, November). *Fetal Alcohol Syndrome/Fetal Alcohol Effects: Action Plan*. Whitehorse: Author.

Appendix A: Research Limitations

The complexity in researching ARBDs has resulted in diverse, sometimes contradictory findings. In part, the inconsistencies are due to various methodological difficulties encountered in this area. To assist in assessing the validity and usefulness of the research, some of the limitations in the ARBD literature are highlighted below.

Prospective versus retrospective studies have been conducted in this area and have often been used to estimate the incidence of FAS. Both types of studies have certain biases that may result in erroneous estimates of FAS. Retrospective studies involve a review of past records—reviewing past records for cases of children diagnosed as FAS, for instance. Methodologically, this type of study is less rigorous than prospective studies (Abel & Sokol, 1991). Such studies are less able to control or examine other factors, such as environmental effects or selection bias, that may account for the findings (Coles et al., 1991). For example, after several years, it is difficult to reliably measure a mother's pattern of prenatal alcohol consumption. Retrospective studies typically over-identify problems associated with a specific cause (Coles et al., 1991). The diagnostic criteria used to identify cases may be distorted and result in an overestimate of cases.

Prospective studies usually involve selecting a sample of pregnant women at a clinic or institution and prospectively following the children born to the sample of women beyond infancy (Coles et al., 1991). One difficulty with this approach is that women with the greatest risk for FAS and other ARBDs may not be included in this type of study because often they do not receive prenatal care and therefore

would not be recruited (Abel & Sokol, 1991). Another difficulty with prospective studies is sample attrition. It is not unusual to encounter problems locating people and keeping their interest in participating when a study is conducted over several years.

Initially, when FAS was first identified, only retrospective studies were possible. Follow-up assessments of FAS patients were primarily based on case studies of children diagnosed with FAS at birth—these studies suffered from small sample sizes and unrepresentative samples of the larger population (Day & Richardson, 1991). More recently, several prospective studies have been conducted that are based on substantial sample sizes (Day et al., 1991; Olson et al., 1992).

The accurate measurement of maternal alcohol consumption in research studies is very important as the information is used to identify the “light,” “moderate” and “heavy” drinkers in samples, and research findings comparing outcome (e.g., number of children born with FAS) for each of these subgroups relies heavily on the validity of these data. However, because research studies cannot measure actual alcohol consumption during pregnancy, they rely on mothers' self-reports of alcohol use.

Retrospective studies in particular are subject to problems in ascertaining accurate estimates of a woman's alcohol consumption during pregnancy. Because information concerning the amount and frequency of alcohol ingested prior to and during pregnancy is often requested months or even years after the birth, it is extremely difficult for women to accurately recall their intake. Even more carefully controlled prospective studies that collect alcohol consumption data throughout the pregnancy generally suffer from underreporting. In general, most abusive drinkers tend to underreport

their alcohol intake due to denial or an inability to recall intake (Sokol, 1981). Ernhart (1991) also reports that most women do decrease their levels of consumption during pregnancy, making quantifying the amount consumed even more complex and difficult.

An additional problem encountered when collecting maternal alcohol consumption information is that when women are asked to recall their first trimester alcohol use, they often do not consider their intake prior to recognition of the pregnancy (Day & Richardson, 1991). Some studies have attempted to counteract this problem by asking women to discuss their drinking behaviour immediately prior to pregnancy, in the belief that this will capture the period immediately after conception (Hanson et al., 1979 as cited in Jones, 1986).

There are numerous difficulties in diagnosing ARBDs such as FAS which can result in biased research results. The reasons for the difficulty in making a diagnosis are discussed in more detail in Section 3.2 (Difficulties Diagnosing Fetal Alcohol Syndrome). However, briefly, they are:

- there is no single symptom distinctive of FAS;
- there are no standardized diagnostic tools for FAS;
- many symptoms vary with age;
- facial features are difficult to recognize;
- the validity of using some diagnostic criteria cross-culturally is questionable (e.g., facial characteristics, IQ tests); and
- it is often overlooked because it is a relatively newly identified syndrome.

Identifying children with alcohol-related birth defects other than FAS is even more difficult. Many children with ARBDs do not exhibit

the physical characteristics associated with FAS, and many have normal or near-normal IQs.

An objective evaluation of the epidemiological evidence is important when attempting to answer such questions as: "Is the incidence of FAS higher in the Aboriginal population?". As Bray and Anderson (1989) point out, there is a lack of FAS statistics in Canada for non-Aboriginal and Aboriginal populations. Canadian FAS incidence rates are usually based on U.S. incidence estimates. The lack of Canadian statistics makes it difficult to assess the true extent of the problem in Canada. Canadian incidence estimates are discussed in Section 7.2 (Incidence in Canada).

The results of some research studies need to be interpreted with caution as they have not adequately controlled for confounding variables (i.e., other than alcohol consumption) that may account for the birth defects under consideration. Day and Richardson (1991) state: "One problem in interpreting these follow-up data lies in the difficulty of separating the teratogenic effects of alcohol from the effects of the disordered environments, both interpersonal and structural, that often accompany alcohol and drug use."

Many factors are cited to complicate the effects of drug and alcohol exposure, including general health, nutrition, housing, income and tobacco (Addiction Research Foundation, 1992). Other sociodemographic variables that are independent risk factors for problems during pregnancy, and thus need to be carefully measured when doing research, include: race, education, income, marital status, adequacy of prenatal care, and use of tobacco, marijuana and other illicit substances (Day & Richardson, 1991).

Appendix B: The Physical Effects of Alcohol on the Fetus

Based on a comprehensive review of case study and epidemiological literature, Abel (1990) summarizes the frequency of occurrence of various types of alcohol-related birth defects. The following discussion is primarily based on Abel's (1990) text and presents an overview of the incidence of anomalies that occur prior to, during, and following birth.

Problems encountered during pregnancy or birth

Spontaneous Abortion Rates

Abel (1990) concludes that alcohol consumption during pregnancy increases the risk of spontaneous abortion only for heavy drinkers. He cites a study by Sokol in 1985 which found that the single factor related to spontaneous abortion among alcoholic women was an extremely heavy episode of drinking during the early first trimester.

Stillbirths

According to Abel (1990), the majority of studies fail to find a statistically significant increased rate of stillbirth among alcoholic women. But most reported still include it as a possible result.

Prematurity

Most studies show no significant increase in preterm births for women who drink less than six drinks per day. The nature of the relationship between maternal alcohol consumption and prematurity is still unsettled. Where there is evidence, prematurity may be due more to malformations than to alcohol ingestion itself.

Breech Births

Abel (1990) concludes that Fetal Alcohol Syndrome is definitely associated with breech presentation—35% of FAS children are born breech. That figure would be 70% if cesarean section deliveries were considered.

Problems encountered after birth

Prenatal Growth Retardation

Low birth weight is one of the key diagnostic features of FAS, though some studies find that when birth weight has been adjusted for gestational age, maternal alcohol consumption itself in these studies appears to account for no more than 2% of the decrease relative to average birthweight. Possibly then, low birth weight is actually an indication of prematurity, rather than a result of true growth retardation. Some studies also report statistically significant decreases in birth weight for moderate or light drinkers; however, Abel (1990) considers these decreases to be well within normal range when other risk factors are taken into consideration.

Postnatal Growth Retardation

Studies indicate that FAS children do not display "catch up" growth at least until puberty and Streissguth (1988) states that short stature remains into adulthood.

Craniofacial Effects

In 1985, Clarren et al. (as cited in Abel, 1990) reported that the main facial characteristics in FAS individuals were: short palpebral fissures, a long midface relative to normal nasal length, a long flat philtrum, a thin upper lip and a flat midface. Clarren et al. (1987) (as cited in Abel, 1990) found that craniofacial effects are more numerous and obvious in children whose mothers drank substantial

amounts of alcohol—facial features are much less distinct in the offspring of moderate drinkers. It has also been observed (Phillipson, 1988; Overholser, 1990) that those individuals with the most apparent craniofacial abnormalities tend to have the most severe microcephaly, shortest stature and most impaired intellectual functioning.

Anomalies of the eyes

The various anomalies include:

- short palpebral fissure size (the size of the opening between the eyelids) appears to be a subjective rather than objective diagnosis; incidence of this anomaly in the literature ranges from 11% to 90% according to Abel (1990). It is important that race-related normative data be used when evaluating palpebral fissure size;
- the incidence of an epicanthic fold (a vertical fold of skin on either side of the nose, near the inner eye) in FAS patients ranges from 9% to 68%;
- strabismus (crossed eyes) rates range from 20% to 56% in various studies;
- antimongoloid slanting (opposite to that characteristic of Down's Syndrome) of the palpebral fissures and hypo/hypertelorism (decreased distance between the two eyes) are less common—37% according to Majewski and Goecke (1982) (as cited in Abel, 1990);
- microphthalmia (abnormal smallness of the eyeball), which is often singled out as a characteristic FAS facial feature, is much less prominent than some of the aforementioned features;
- visual disorders appear to be quite common though most FAS patients are not specifically examined for these anomalies. Myopia (near-sightedness) is most common. Stromland (1985) (as cited in Abel, 1990) found 23% of children had such poor vision that they were visually handicapped. It is possible that visual disorders may account for poor fine motor dysfunction and perceptual difficulties;
- retinal tortuosity (curves or twisting of the retina) ranges from 10% to 94% in the literature;
- optic nerve anomalies are also present; and
- corneal astigmatism (abnormal curvatures in the cornea) appears to be particularly common (91%) among Navajo Indian school children though this has not been verified with other subgroups.

Anomalies of the Ear

The various anomalies include:

- outer ear anomalies are often mentioned (22% to 59%);
- inner ear anomalies and hearing problems appear to be quite common. Abel (1990) believes that all FAS children should be tested for hearing problems at an early age;
- sensorineural hearing loss is found in 29% of FAS children; and
- one thorough study found that 13/14 FAS children had bilateral recurrent serous otitis media (inner ear infections). This affliction is associated with eustachian tube dysfunction and is related to anomalies of the first and second bronchial arches which can also result in low-set ears, abnormal ears, and cleft lip and palate.

Oral Features

The various anomalies include:

- micrognathia (abnormal smallness of the jaw) is commonly reported as is cleft palate, high arched palate, and narrow vermillion (border of the upper lip); and
- dental problems are also likely as maxillofacial (bones forming the upper jaw) anomalies are common.

Organ Pathology

Pathology of vital organs is very common and should have been included in the standard FAS diagnostic criteria according to Abel (1990). Cardiac problems are reported in 18% of

the case reports that Abel (1990) reviewed in the literature. Most common were ventricular and septal defects. Liver anomalies were rare. Urogenital anomalies occurred in about 10% of FAS cases. Genital anomalies were noted in 12% of cases, while inguinal hernia (hernia occurring in the groin) was found in 3.5% of FAS patients.

Skeletal Defects

Limb, joint and other skeletal problems were cited in 18% of FAS cases. Clinodactyly (deviation of one or more fingers) and camptodactyly (a claw-like condition of the hand or foot) were commonly reported though hypoplastic nails (incomplete development of nails) may be even more common. Other problems included areas of calcium deposits, missing limbs, radial ulnar fusions, missing bones, hip dislocation, scoliosis and club-foot. If limited flexion and other movement problems were included as skeletal defects, the proportion of FAS patients afflicted with skeletal defects would have been much higher than 18%.

Neural Tube Defects

Incidence rates range from 18%-35% for neural tube defects.

Immunological Function

FAS patients have fewer lymphocytes and increased eosinophil counts suggestive of allergic reaction or altered susceptibility to infections. As previously mentioned, recurrent otitis media is most common. A recent review of human and animal research to assess alcohol's effect on fetus' immune systems found that immune systems definitely are impaired (though the mechanisms responsible for producing the damage have not yet been identified) and the effects on the

immune system are long-lasting (Giberson & Weinberg, 1992).

Obstruction of Upper Airway Passages

As craniofacial anomalies are common in FAS, obstruction of upper airway passages is likely more common than presently recognized. Mandibular hypoplasia (incomplete development of the horseshoe-shaped bone forming the lower jaw), that is common in FAS, may also cause respiratory distress and stridor (particularly at night). Upper airway obstruction in FAS patients may also contribute to Sudden Infant Death Syndrome (SIDS). According to Southall et al. (1987) (as cited in Abel, 1990), mothers of SIDS babies drank more often than mothers of babies who did not succumb to SIDS.

Neuropathology

Microcephaly (small head size), indicating underdevelopment of the brain, is common; absence or under-development of the corpus callosum (fibers connecting the left and right halves of the brain) and enlarged lateral ventricles (fluid filled chambers) are most commonly noted.

Tumors

As is the case with most teratogens, exposure to alcohol late in pregnancy may result in tumor generation. However, no common type of tumor is found in FAS children. In those instances where tumors have been discovered, they were found before the child was two years of age and many of the mothers were using anticonvulsant medication. The tumors could have been due to the anticonvulsant medication taken during pregnancy or its interaction with alcohol during pregnancy.

Central nervous system (CNS) Involvement

The link between prenatal alcohol exposure and later mental retardation and sensory deficits are generally detectable only when the child enters school.

- Sleep disturbances in the newborn are commonly reported they have trouble reaching quiet sleep, are more easily awakened, more restless and have more gross body movements.
 - Electroencephalography (EEG) hypersynchrony (abnormal EEG activity or brain wave activity) is as much as 200% higher in FAS infants and led Ioffe et al. (1984) (as cited in Abel, 1990) to propose that "the current definition of FAS, which is based on dysmorphology, may be inadequate because EEG differences clearly are seen in the absence of dysmorphology and may be a sensitive indicator of fetal alcohol toxicity."
 - Increased seizure/convulsion activity was one of the earliest CNS effects noted; 3.3% of FAS patients recorded in various studies had such problems.
 - An increased incidence of cerebral palsy in FAS individuals has been suggested by Ollegard (1979) and Lipson et al. (1983) (as cited in Abel, 1990).
 - A weak sucking reflex is noted in many infants and may contribute to their failure to thrive.
 - Speech and language – FAS children have significantly higher incidence of speech impairment, reduced vocabulary and clarity of speech. Speech delay and impediments may be partially due to hearing problems or disorders of the palate.
-

Appendix C: How Alcohol is Processed by the Fetus

When the mother drinks, the alcohol consumed passes directly through her stomach wall and enters her bloodstream. Alcohol gets into the bloodstream of the fetus by crossing the thin membrane of the placenta. At the time of transmission across the placenta, fetal blood alcohol levels approximate those found in the mother (Streissguth, 1980, & Rose, 1981 as cited in Overholser, 1990). However, the fetal blood alcohol concentration remains elevated for a longer period of time because the fetal liver is underdeveloped and is not able to break down the alcohol as rapidly as the mother's. It is estimated that newborns metabolize alcohol at half the rate of an adult (Idanpaan-Heikkila, 1972 as cited in Abel, 1990). Once the maternal blood alcohol concentration falls lower than the fetal blood alcohol level, the alcohol begins to diffuse back to the mother's bloodstream via the placenta.

Due to the nature of the biological processes involved, it is apparent that if the mother drinks heavily on a regular basis, a consistently high blood alcohol concentration remains in both the mother and in the fetus. This continuing exposure leads to the fetus becoming physically dependent on alcohol. Withdrawal symptoms have been seen in newborns from birth to 12 hours after birth (Robe et al., 1981, & Coles, 1984 as cited in Abel, 1990).

In addition to the toxic effects of the alcohol itself, alcohol consumption appears to be related to many other physiological disturbances in the fetus. Alcohol inhibits the mother's absorption of nutrients into the bloodstream and, by disrupting hormones, lessens the ability of the placenta to transport nutrients to the developing baby (Abel &

Greizersten, 1982; Lin, 1980; Fisher, et al., 1981, all as cited in Overholser, 1990). Alcohol also has been shown to alter the DNA⁴ and RNA⁵ of the fetus (Thadani, 1981 as cited in Overholser, 1990), disrupt growth hormone secretion (Thadani, 1980; Rawat, 1981; Redmond, 1981, all as cited in Overholser, 1990), and can suppress immune system activity which increases susceptibility to infection (Heine, 1980 as cited in Overholser, 1990). Structural anomalies in the brain due to retarded CNS myelination also appear to result from alcohol ingestion during pregnancy (Boggan, 1982 as cited in Overholser, 1990).

⁴ Refers to the genetic material deoxyribonucleic acid (Miller & Keane, 1972).

⁵ Refers to the genetic material ribonucleic acid (Miller & Keane, 1972).

Appendix to the Report on the State of the Nation

The following table shows the results of the various surveys conducted by the Department of the Interior during the year ending June 30, 1898. The figures are given in thousands of acres, unless otherwise stated.

Category	Area (in thousands of acres)
Public Lands	1,234,567
Indian Reservations	456,789
Forest Reservations	234,567
Mineral Reservations	123,456
Other Reservations	98,765
Total	2,148,184

The above table shows the results of the various surveys conducted by the Department of the Interior during the year ending June 30, 1898. The figures are given in thousands of acres, unless otherwise stated.

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